A STUDY ON ETIOLOGY, CLINICAL COURSE AND VISUAL OUTCOME OF RETINAL VASCULITIS

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CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON ETIOLOGY, CLINICAL COURSE AND VISUAL OUTCOME OF RETINAL VASCULITIS"** presented herewith by ***Dr. R. SARAVANAN*** to the faculty of Ophthalmology, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. degree in Ophthalmology is a bonafide work carried out by him under my direct supervision and guidance.

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CONTENTS

INTRODUCTION

Retinal Vasculitis is a non specific immunologic reaction of retinal arterioles or venules to various antigenic stimuli that may occur in many different systemic and local disease states.

It may be defined, however, to be present when there are vascular leakage and staining of vessel walls on fundus fluorescein angiography, with or without clinical appearance of fluffy, white perivascular infiltrates in an eye with evidence of inflammatory cells in the vitreous body or aqueous humor.

Retinal vasculitis may or may not be symptomatic. Inflammation of peripheral retinal blood vessels is typically asymptomatic, but inflammation involving macular retinal blood vessels frequently causes decreased visual acuity and focal scotomas. Associated vitreous inflammatory cells can give rise to floaters.

The signs of vascular sheathing or cuffing can be found in inflammatory vascular disorders as well as in ischemic vasculopathies without inflammation. These two conditions should not be confused, as the approach to the diagnosis is different. If vascular sheathing is accompanied by vitreous cells, adjacent intra retinal edema, or infiltrates, it is more likely to be inflammatory in orgin.

Fundus fluorescein angiography is useful in evaluating the severity and extent of disease. Mildly inflamed vessels usually only show staining with fluorescein, whereas in more severe inflammation, dye leakage occurs as well. In occlusive vasculitis, capillary fallout is seen. If untreated, neovascularization, vitreous hemorrhage, and tractional retinal detachment may ensue[51].

History

In 1887 Wads Worth was first to describe the clinical picture of retinal periphlebitis, 1873 Perls performed histological examination of periphlebitis in case of T.B. In 1880 Henry Eales described the syndrome and he associated epistaxis and constipation with recurrent Vitreous hemorrhage in later paper, excluded known systemic disease including, diabetes, clotting abnormalities, blood dyscariasis, syphilis. In beginning of early century relation with T.B. suggested.

Duke – Elder regarded Eales disease as clinical manifestation of inflammatory retinal vessel occlusion or periphlebitis. Eales disease occurs frequently in India finding difficult to distinguish this condition from other type of retinal Vasculitis.

DISORDERS ASSOCIATED WITH RETINAL VASCULITIS[26] (CLASSIFICATION)

I. Infections Associated with Retinal Vasculitis

Tuberculosis, Syphilis, Borreliosis – Toxoplasmosis, Herpes virus, Cytomegalo Virus, Acute Retinal Necrosis, Candidiasis, Leptospiroisis, Brucellosis, Cat scratch disease, Rickettsia, Epstein Bar virus, Whipple's disease, HIV, Rift valley fever virus.

II. Vasculitis secondary to systemic Autoimmune disease

Behcet Disease, Crohns Disease, Rheumotaid Disease Buerger's disease, Sjogren's syndrome A antigen. HLA B27 associated uveitis Sarcoidosis,

III. Retinal Vasculopathy in systemic Vasculitis

Systemic Lupus Erythematosus, Wegeners Granulomatosis, Poly Arteritis Nodosa,

IV. Vasculitis secondary to Ocular disorder

1. Eales disease, Idiopathic retinal Vasculitis, pars planitis, bilateral iridocyclitis with retinal capillaritis, frosted branch angitis, bird shot retinochoroidopathy, Idiopathic retinal Vasculitis aneurysm and Neuroretinitis, Idiopathic recurrent branch. Retinal Arteriolar

Occlusion, Sympathetic Ophthalmia, Primary ocular disease, Vogt – Koyanagi – Harada Syndrome

V. Retinal Vasculitis Associated with Neurologoic Disorders

Multiple Sclerosis, Isolated central Nervous system angitis, Micro Angiopathic encephalopathy, Hearing loss and Retinal arteriolar occlusion isolated central nervous system angitis.

VI. Drug Induced – Retinal Vasculitis

VII. Retinal Vasculitis secondary to malignancy

VIII. Miscellaneous cause of Retinal Vaculitis

Retinal vascular anatomy

There are two sources of blood supply to the retina: the central retinal artery and the choroidal blood vessels. The choroid receives the greatest blood flow (65-85%) (Henkind et al., 1979) and is vital for the maintenance of the outer retina (particularly the photoreceptors) and the remaining 20-30% flows to the retina through the central retinal artery from the optic nerve head to nourish the inner retinal layers. The central retinal artery has 4 main branches in the human retina

The arterial intraretinal branches then supply three layers of capillary networks i.e. 1) the radial peripapillary capillaries (RPCs) and 2) an inner and 3) an outer layer of capillaries. The precapillary venules drain into venules and through the corresponding venous system to the central retinal vein

The radial peripapillary capillaries (RPCs) are the most superfical layer of capillaries lying in the inner part of the nerve fiber layer, and run along the paths of the major superotemporal and inferotemporal vessels 4-5 mm from the optic disk (Zhang, 1994). The RPCs anatomose with each other and the deeper capillaries. The inner capillaries lie in the ganglion cell layers under and parallel to the RPCs. The outer capillary network runs from the inner plexiform layer to the outer plexiform layer through the inner nuclear layer (Zhang, 1974).

A ring of blood vessels in the macular area around a blood vessel and capillary-free zone 450-600mm in diameter, denoting the fovea. The macular vessels arise from branches of the superior temporal and inferotemporal arteries. At the border of the avascular zone, the capillaries become two layered and finally join as a single layered ring. The collecting venules are deeper (posterior) to the arterioles and drain blood flow back into the main veins.

It is worth identifying retinal vessel to correlate the retinal features of systemic Immune diseases with size of vessels involved[42].

Vessel involved	Disease	Retinal Features
Muscular arteries	Poly arteritis Nodosa	Cotton wool spots, retinal edema, Hypertensive changes
Medium to small arteries	Wegeners granulomatosis	Cotton wool spots, arteriolar occlusion
Small arteries	Systemic lupus erythematosus	Cotton wool spots, retinal edema, Haemorrhage
Capillaries	Whipple's disease	Haemorrhage, Exudate, capillary occlusion
Veins	Behcet's disease Sarcoidosis Multiple sclerosis	Infiltrate branch retinal vein occlusion, macular edema neovascularization Periphlebitis, venous sheathing, choroidal Granuloma, neo vascularization. Venous sheathing, macular edema, neovascularization
Arteries and Veins	Crohn's disease Poly chondritis	Vascular occlusion Exudate with Haemorrhage
Miscellaneous	Ankylosing spondylitis HLA – B 27 positive	Macular edema, vitritis, diffuse micro vascular leakage

PATHO PHYSIOLOGY AND IMMUNOLOGY

RETINAL VASCULITIS IS BELIEVED AS IMMUNOLOGICAL MEDIATED CONDITION[26]

Retina contain number of tissue-specific antigen, one of which Retina-S-antigen. Ocular inflammation induced by S-antigen is marked retinal Vasculitis accompanied by focal mononuclear cell infiltrates and necrosis of photo receptor cell layer. Patient have systemic inflammatory disease could develop retinal Vasculitis because of presence of circulating immune complex, non organ specific auto immune or abnormal white cell functions. Isolated retinal Vasculitis may be an organ specific auto immunological disease of retina.

MAJOR COMPONENT IS CELL MEDIATED IMMUNITY

Presents of CD4+ T cell as predominant cell in vascular lesion of eye, supports assumption of retinal Vasculitis is cell mediated immunity.

(a) Adhesion – Adhesion interaction molecule (selection, integrin) enables leukocyte adherence to vascular endothelium (Carbohydrate). Chemokine also plays an important role.

(b) Tethering and rolling – Activated endothelium to express P-selecting and E-selecting that interacts with carbohydrate moieties of leukocyte – slow their speed and roles on endothelium.

(c) Latching and activation – Lymphocyte function-associated adhesion molecule 1(LFA-1) binds to endothelial cell ligand called inter cellular adhesion molecule (ICAM-I, II, III)-gives an stronger attachment. Other antigens are very late antigen (VLA) with vascular cell adhesion molecule (VCAM).

(d) Migration – activated leukocytes flatten to the endothelium, affinity of leukocyte integrin is unregulated and expression of late binding protein occurs it initiates migration through endothelium and basement membrane to from peri vascular cuff of inflammatory cells.

HUMORAL IMMUNITY AND IMMUNE COMPLEX: are still controversy

Although type III hyper sensitivity is a mechanism of tissue damage is systemic vasculitis.

On presence of dominating CD4+, T cell respond retinal vasculitis may also have a humoral response and this may have an Immune regulatory function perhaps influenced by Immune complex formation.

SYMPTOMS AND SIGNS[10,18,46]

1. Symptoms:
    - Inflammation of peripheral vessels may be completely asymptomatic
    - Painless blurring or loss of vision – due to vitreous haemorrhage
    - Large Scotoma corresponds to area of ischemia
    - Floaters
    - In eales disease sudden loss of vision to vitreous hemorrhage.
    - Sign of systemic involvement like Oro-genital ulcer, Arthritis, skin rash, respiratory and neurological symptoms
2. Signs:

    (a) Anterior Uveitis can be seen in 1/3rd of cases

    1. Circumcorneal (ciliary) congestion – in associated with glaucoma
    2. Keratic precipitates (a) Endothelial dusting
        (b) Medium –size Keratic precipitates
        (c) Large Keratic precipitates
        (d) Old Keratic precipitates
    3. Cells are indicative of active inflammation
        (a) Aqueous cells

(b) Anterior vitreous cells

4. Aqueous flare-faint: just detectable = + 1

   Moderate: Iris details clear = + 2

   Marked Iris details Hazy = + 3

   Intense with Fibrinous exudates= + 4

5. Iris nodules-are features of granulomatous inflammation. Koeppe's nodule and Bussaca nodule.

   (b) Cellular infiltrations of vitreous as seen in all cases.

   (c) Inflammatory cells or snow balls in inferior vitreous cavity.

HALL MARK – VASCULAR CHANGES

In active phase-Sheathing of vessel, fluffy white cuffing around vessel throughout vessel or like skip lesion, sheathing most often in veins then arterioles.

In chronic phase-sheathing seen in peripheral retina and not associate with flourescein leakage from periphlebitis.

An extreme form of exaggerated sheathing of vessels gives of clinical picture that resemble tree branches in winter called FROSTED BRANCH ANGITIS.

In some conditions like sarcoidosis discrete waxy nodule are seen on the retinal vessel, in human T cell lymphoma virus type 1 (HTLV 1) – characteristic gray white granular deposit on retinal vessel.

In addition to vascular changes retinal edema is common and may proceed vascular cuffing, deep retinal infiltrate in some patient.

In Retinal-vascular occlusion associated with Behcets disease leads to retinal hemorrhage, Cotton wool spot, pallor in areas of retinal Ischemia. Vessels in these areas become sclerotic attenuated. Retinal Neovascularisation in 16 – 40% cases of retinal vasculitis along with vitreous hemorrhage.

RETINAL VASCULITIS IN OCULAR DISEASE

IDIOPATHIC RETINAL VASCULITIS[31,49,61]

Occurs in young adults and causes blindness

Ophthalmoscope feature shows sheathing of retinal vessels and vitritis.

Fundus flourescein angiography reveal areas of clinically invisible disease, confirms the diagnosis in the absence of systemic condition finding by history and examination. Full blood count, sedimentation rate SEROLOGY FOR SYPHILLIS, X-RAY CHEST.

Visual outcome variable and depends on presence of retinal ischemia. Ischaemic group having vision of 6/60 or worse on compare with non ischemia. In a retrospective study, 59% chance of developing systemic disease, major event is cerebro vascular accident. So blood pressure and lipid profile are important and also history of smoking.

EALE'S DISEASE[12,20]

Henry Eales (1880-1882) was the first to describe a clinical syndrome of recurrent retinal and vitreous hemorrhages in apparently healthy young men.

Classifically manifest as obliterative periphlebitis anterior to equator involving multiple quadrants with progression posteriorly. Eales stimulate as

Idiopathic Branch Retinal Vein Occlusion do not occur at AV crossing site and may / may not be associated with Cotton Wool Spot.

CLINICAL PRESENTATION

Classically the presenting feature of the Eales disease is repeated vitreous hemorrhage patients compliant of sudden appearance of floating spots, cobwebs, cloudy vision, or simply reduced vision. Vision often improves with time before a repeat hemorrhage affects vision again. The recovery of vision usually becomes less and less complete with recurrent episodes. Finally, the patient may be left with no useful vision. The gross loss of vision may be due to unresolving vitreous hemorrhage or such complications as traction retinal or macular detachments. Often a routine examination of the contra lateral eye reveals evidence of present or past phlebitis. A normal contra lateral eye, in the course of time, may develop the disease. At the time to presentation the contra lateral eye was found to be affected in more than 50% of cases. Ultimately, the involvement becomes bilateral in 80% to 90% of cases.

All patients do not present in this classical form, however. Some present themselves with sudden gross loss of vision that does not clear for years, the result of massive vitreous hemorrhage during the initial stage of acute phlebitis. Others complain of reduced or blurred vision because of macular edema, hemorrhages, or concomitant macular infarction.

A sizeable number of patients are not aware of the disease because the lesions are peripheral. The signs of disease are noted during routine fundus examinations. Although phlebitis is less common among women, when it does appear it is usually much more severe and difficult to control, and the post-treatment results are less satisfactory. Although one often observes uveitis (Choroiditis), papillitis, or Arteritis associated with phlebitis, it is phlebitis that is central to the clinical manifestation of Eales disease.

PATHOPHYSIOLOGY

The basic pathology of Eales disease is retinal phlebitis multiple branches of central retinal vein are inflamed, causing multiple inflammatory BRVOs. Occassionally, the central retinal vein may be inflamed and occluded with or without associated inflammation of branch retinal veins, resulting in inflammatory CRVO. A CRVO in a young adult man who is neither hypertensive nor diabetic, and who has no signs of arteriosclerosis, would suggest phlebitis. Dramatic response to oral corticosteroids could help confirm the diagnosis. The severity and the course of phlebitis vary widely.

ACUTE COURSE

An acute and rapid course results in sudden and complete occlusion of the retinal veins, which could lead to subhyaloid or vitreous hemorrhage. When a large-caliber retinal branch vein is suddenly occluded, a massive vitreous

hemorrhage results that does not clear for months or years. The diagnosis is made on the basis of findings in the contra lateral eye and by excluding other causes. On the other hand, when a smaller caliber peripheral venule hemorrhage is relatively less massive and clears with time. Occasionally, more than one peripheral venule becomes involved in sequence, causing RVH during stages of acute phlebitis.

NONACUTE COURSE

Here phlebitis can be very mild and chronic, causing little or no noticeable vascular decompensation in the form of retinal hemorrhage or retinal edema.

SUBACUTE COURSE

Most cases fall into this category. The retinal sector being drained by the inflamed branch shows retinal hemorrhages and edema. There could be cotton wool spots and dark blot hemorrhages, indicating retinal ischemia. Peripheral, segmental ischemia owing to inflammatory BRVO leads to NVE originating just proximal to the site of inflammation and occlusion. In cases with multiple venous branch involvement and extensive peripheral ischemia one would expect NVD along with multiple peripheral NVE. Often one finds the classical sea-fan neovascular lesion in the retinal periphery.

Because retinal ischemia in cases of multiple BRVO can be well defined and limited to the territories of venous drainage, selective retinal ablation by scatter photocoagulation or cryoablation need to be restricted to these defined areas. Indiscriminate panretinal photocoagulation has no place in the management of Eales disease, even in cases with NVD.

Some areas show marked capillary engorgement, which sometimes leads to small RVH. Sometimes one can demonstrate multiple microaneurysms.

The inflamed veins present varied pictures. There could be massive exudation over large tracts of the vein, causing complete occlusion of vein segments. The vitreous may be turbid. When inflammation subsides, some of the veins would show complete destruction of the vein segment on FA. Some cases show nodular formation over the inflamed veins. These cases may be inflamed veins. These cases may be presumed to be cases of tubercular phlebitis.

Drugs for tuberculosis have been found to be effective. In less severe cases, variable degrees of infiltration in and around the veins in segmental fashion can be seen. These segments, during the stage of active inflammation, show a pale white, fluffy, finely irregular outline. Fluorescein angiography shows delayed filling, prolonged circulation time, and a narrowed lumen. In the

late phases, the inflamed vein segments may show dye staining and even dye leakage through the venous walls.

On resolution of phlebitis, the inflamed segments show parallel sheathing, kinking, or looping owing to gliosis. Often one finds choriorentinal scars under the inflamed segment. The chorioretinits seems to be secondary to phlebitis. Even on sometimes demonstrate delayed circulation in the area involved.

Healing and the Process of Circulatory Compensation

Phlebitis subsides with time. Treatment with anti inflammatory and other drugs accelerates the process. The venous obstruction tends to get relieved, with the lumen opening up to a variable degree. Some acute cases, however, show permanent destruction of vein segments. Veno-venous capillary develop from obstructed to normal territory. These capillaries dilate and show tortuosity, one of the most common findings in Eales disease. Occasionally the dilated capillaries shows macro aneurysm that could leak fluorescein. Rarely one may see large shunt vessels.

The retina with compromised circulation and ischemia gradually becomes atrophic, with reduced circulation and reduced venous return. The arterioles look reduced in caliber and the venous engorgement disappears. The

stimulus for vascular proliferation disappears. The new vessels, which had developed in response to retinal ischemia, now tend to regress with reduced vascularity.

The regressing new vessels lead to glial proliferation which, in turn, causes several serious complications. The contracting glial tissue may detach or distort the macula and cause macular degeneration, macular cyst, or macular holes. These may cause traction retinal tears leading to rhegmatogenous retinal detachment. Sometimes, the macula may get covered with thick glial tissue, compromising vision.

What happens as a natural course of the proliferative retinopathy is achieved through uncontrolled selective retinal ablation by scatter photocoagulation and retinal cryoablation. Anchoring the neovascular lesions to the underlying retina and choroids by strategically placed photocoagulation marks, before the neovascular lesions are made to regress by scatter photocoagulation, would avoid most of the traction related complications.

INVESTIGATIONS

Flourescein Angiography

Valuable in revealing the degree of retinal capillary Non perfusion and presence of Neovascularisation as a guide to laser.

The inflamed venous walls stain with narrowed lumen and may show some swelling and dilation distal to the site of inflammation or even frank extravasation. On the other hand, parallel sheathing, caused by gliosis after the inflammation has subsided, does not show this feature. In most cases, the segmental venous inflammation produced varying degrees of venous occlusion. The lumen may be narrowed or even closed.

Fluorescein angiography study the course of this vascular disease both structurally and functionally. Although cases of proliferative retinopathy are being treated by selective retinal ablation with scatter photocoagulation and cryoabalation, the regression of new vessels can be monitored with extreme precision by Fluorescein Angiography.

PARS PLANITIS

An intermediate Uveitis, usually bilateral and varies from mild inflammation to blinding condition Ocular manifestation include variable number of spill over anterior chamber cells, Vitreous cells, snow balls, and snow banks. Inferior peripheral retinal phlebitis with retinal venous sheathing is common with chronic inflammation cystoid macular edema develops. Ischemia from retinal phlebitis, combined with angiogenic stimuli from intraocular inflammation can lead to neovascularization, results in vitreous haemorrhage, contract and lead to peripheral tractional and rhegmatogenous retinal detachment.

Differential diagnosis: syphilis, lyme uveitis, sarcoidosis

Treatment: Should be directed to wards treating the underlying cause of inflammation if possible. Treatment is usually implemented if visual acuity 6/12 or worse or if cystoid macular edema present. Otherwise no treatment required

Step I: Periocular corticosteroid constitute first line of therapy.

Triamcinolone / methyl prednisolone may be used. Injections are usually repeated every 2-3weeks. Until four injections have been administered.

If local not effective, systemic steroids were used, and reserved for more severe or bilateral cases.

Step II: If corticosteroid fails peripheral ablation of the pars plana snow bank with cryo theraphy or indirect laser photo coagulation should be performed.

Step III: If cryo theraphy fails and systemic immuno suppressive is contraindicated or not desired pars plana vitrectomy with induction of posterior hyaloid separation and peripheral laser photocoagulation to pars plana snow bank performed.

Step IV: If all other therapeutic failed systemic immuno modulating agents are used.

IDIOPATHIC RECURRENT BRANCH RETINAL ARTERIOLAR OCCLUSION

Common in middle age groups recurrent branch retinal arteriolar occlusion. No inflammatory disease or evidence of embolism

Focal perivascular sheathing and flourescein angiography shows multiple segment arteriole staining. Pre retinal neovascularisation occurs in area of ischemia. No systemic cause found but 50% patient has vestibule or transient sensory motor symptoms or both.

PRIMARILY OCULAR DISEASE

In bird shot retinopathy retinal vasculitis of venules is a prominent and consistent feature. It may present as idiopathic retinal vasculitis with the development of characteristic focal cream coloured lesion.

RETINAL VASCULITIS AND NEUROLOGICAL DISEASE

Multiple sclerosis[26]

Ocular inflammatory signs in multiple sclerosis are iritis, intermediate uveitis, posterior uveitis, periphlebitis, optic neuritis. In most cases periphlebitis is subtle peripheral, and of no visual consequence, in some cases leads to occlusive vasculitis, ischemia, retinal neovascularisation.

Patches of fluffy peri vascular cuffing represent areas of active disease, whereas sclerotic whitening of venular wall with no leakage is chronic, periphlebitis due to lympho plasmacytic infiltrate, occasionally with granulomatous component.

25% of retinal vasculitis develop multiple sclerosis particularly if they are female and carry the HLA b27/HLA DR2 alleles.

INFECTIOUS ASSOCIATED WITH RETINAL VASCULITIS

Bacteria

TUBERCULOSIS[51]

Tuberculosis (TB) of the retina is always hematogenously derived from a lesion elsewhere in the body, diagnosed or occult. The most common presentation of retinal involvement is periphlebitis. Isolated retinal periphlebitis is rare, but may be the presenting sign in disseminated tuberculosis. It is often accompanied by a moderate vitreous infiltrate, macular star, and retinal hemorrhage. It may result in branch or central retinal vein occlusion, often with peripheral retinal closure, leading to new vessel formation and vitreous hemorrhage.

Retinal vasculitis has also been reported following a Mantoux test. Tuberculosis retinal vasculitis is not characteristic and may be misdiagnosed unless choroidal tubercles are also present. It should be suspected in the presence of florid retinal vasculitis with marked capillary closure with a relatively mild degree of vitritis. Histology of the retina shows granulomatous lesions surrounding retinal vessels.

The definitive diagnosis of intraocular TB is often difficult, as it requires the identification of Mycobacterium tuberculosis. It is hazardous to obtain

biopsy material from the eye, however, and smears and cultures from aqueous humor or vitreous give a low yield. Polymerase chain reaction can be used to amplify mycobacterial DNA from clinical samples, but it faces the risk of false positive results. A 2-week therapeutic trail of isoniazid is controversial, especially with the increasing prevalence of drug-resistant strains of M. tuberculosis.

A diagnosis of ocular TB, even in the presence of systemic TB, therefore often has to be presumed. Systemic disease may be detected by a chest roentgenogram or positive sputum culture. It is important to have a high index of suspicion in patients with Asiatic origin, those with a history of contact with TB, or when the Mantoux response is strongly positive (usually more than 15 mm induration at 72 hours).

The vasculitis responds to anti-TB therapy, and it should be given in the absence of active systemic disease if tuberculosis ocular disease is strongly suspected, as reactivation of the systemic illness may occur with systemic steroid therapy for retinal vasculitis. Neovascularization of the disc or retinal periphery may require panretinal photocoagulation.

SYPHILIS[51]

Syphilis is caused by the spirochete, Treponema pallidum. It is primarily a sexually transmitted disease, but it can also be spread by transfusion of fresh blood, or by accidental contact with an infected lesion. The ocular manifestations of syphilis are variable and may mimic any from of intraocular inflammation. Retinal vasculitis usually occurs in association with Chorioretinitis, but primary leutic retinal vasculitis is rare and usually involves the arteries with or without occlusion. Isolated periphlebitis and concomitant involvement of artery, vein have also been reported. Leutic vasculitis is commonly associated with severe vitritis, intraretinal and pre retinal hemorrhages, and exudates that are occasionally recurrent or massive. Neovascularization and retinitis proliferans may occur, resulting in narrowing and ensheathing of retinal vessels.

The pathogenic spirochetes cannot be routinely isolated in the laboratory; hence the diagnosis of syphilis is usually based on a history of exposure, clinical findings, and the results of serologic tests. The serologic tests for syphilis are the mainstay of diagnosis when there are no lesions to be examined by direct microscopy. These tests can be divided into two groups:

(1) Those that detect antibody to cardiolipin – lecithin-cholesterol antigen (non treponemal tests), such as Veneral Disease Research Laboratory (VDRL) and RPR.

(2) Those that detect antibody against treponemal antigens, such as FTA – ABS and T. pallidum hemagglutination assay. False-positive results can occur with both types of tests. Nontreponemal tests are best suited for general screening with any positive result confirmed with a treponemal test. Nontreponemal tests are also used to monitor treatment because their titers decrease following effective therapy, whereas FTA – ABS usually remains positive throughout the lifetime of both treated and untreated patients. The serologic tests for syphilis are probably less reliable when co infection with HIV exists. Penicillin G is the drug of choice for treatment is presumptive based on the resolution of clinical findings and the demonstration of a declining serologic titer.

BORRELIOSIS (LYME DISEASE)[51]

Lyme disease is also caused by a spirochete, Borrelia burgdorferi. It is transmitted by the Ixodidae ticks, and patients may develop acute or chronic multisystem inflammation with variable manifestations, including erythema migrans, oligoarthritis, and neurologic and cardiac abnormalities.

Retinal vasculitis may present during both the disseminated and the late stages of the disease. Vessel sheathing and occlusion, intraretinal hemorrhages, vitreous cells, and disc and peripheral neovascularization with vitreous hemorrhage or vitreoretinal traction may develop. Fundus fluorescein angiography may reveal delayed filling of the retinal arterioles and venules with staining of the vessel wall.

Serologic tests, such as ELISA and the immunofluoresence antibody test have limited sensitivity, long latency to response, decreased responsivity after antibiotic treatment, and a cross-reactivity with syphilis and other infections. Therefore, in suspected Lyme disease, VDRL and FTA- ABS should be checked along with Lyme ELISA. A rise in Lyme IgG titers supports the diagnosis of active disease, and may be used to monitor the response to treatment. Polymerase chain reaction testing of the vitreous and CSF may be positive.

The ocular disease responds to antibiotic therapy, such as oral tetracycline in adults, or penicillin V in children. Erythromycin can be used in penicillin allergy. Intravenous ceftriaxone 1 g to 2 g/day for 14 days or longer is recommended for Stage II Lyme disease. The use of corticosteroids is controversial.

BRUCELLOSIS

Brucella infection is transmitted by direct contact with infected animals but mostly by consumption of unpasteurized dairy product.

Uveitis is the most common manifestation. Inflammation may cause, granulomatous / Nongranulomatous anterior uveitis, retinitis, Choroiditis and also retinal vasculitis.

LEPTOSPIROSIS

- Affect eye in both immune and septicemic stages of the disease
- 2week- 1year after infection
- Associated with extensive vitreous membrane periphlebitis, Choroidits, Parsplanitis and disc edema.

PROTOZOAL

TOXO PLASMOSIS[26]

Perivasculitis is caused by arthus – type reaction

- Veins show continuous sheathing over long segment with narrowing of vessels near acute lesions or segmental cuffing of vessels adjacent to active retinitis involved. But sheathing also occur away from the lesion.

- Focal periarterial exudate or plaque –called as kyrieleis arteriolitis are not associated with vascular leakage or obstruction. Vasculitis quickly resolves with resolution of disease and disappearance of antigen.

VIRAL RETINAL VASCULITIS

HIV retinopathy

HIV retinopathy is the most common ocular manifestation, occurring in 40-60% of HIV-positive patients and recognized in 89% of autopsies. Histopathological findings resemble those of diabetic retinopathy, with pericyte necrosis, endothelial cell swelling, and thickening of basement membrane. Hypothesis for vascular injury include immunoglobin deposition, endothelial cell infection by HIV, and hyperviscosity secondary to increased red cell aggregation, fibrinogen, and increased polymorphonuclear leukocyte rigidity. In the anterior segment, these changes are seen as dilated and tortuous segments of vessels.

Occurring in up to 50% of individuals with HIV, cotton wool spots are the most common findings in patients with AIDS. These occur primarily in the posterior pole in the peripapillary region. Cotton wool spots seen in individuals with HIV are identical to those seen in other conditions. Cystoid bodies

represent swollen axons caused by interruption of axoplasmic flow. Although ischemia is the most common cause of cotton wool spots, any interruption of axoplasmic flow leads to the formation of cotton wool spots. Accumulation of axoplasmic debris in the nerve fiber layer leads to the appearance of cotton wool spots.

Retinal hemorrhages develop in up to 30% of patients with AIDS. A variety of hemorrhages are seen, including flame shaped, dot-blot, or peripheral punctate hemorrhages.

Clinically, HIV retinopathy is a sign of a low CD4+ count (usually <50 cells/micro litre) and requires close screening (every 3 months) to look for other manifestations that can occur with such a low CD4+ count, such as cytomegalovirus (CMV) retinitis.

Cytomegalovirus

CMV retinitis is the most common ocular infection in patients with AIDS, occurring in the pre-HAART (highly active antiretroviral therapy) era in 20-40% of patients. The median onset from the diagnosis of AIDS is 9 months, although it has been reported as late as 5 years. The median time to progression

was 47-104 days, with a mean survival after diagnosis of 6-10 months. The risk for CMV retinitis increases with lower CD4+ counts.

Intimate exposure to infectious sources of virus (i.e., blood, urine, saliva, other secretions) sometimes leads to hematogenous seeding of CMV-infected monocytes to the retina. HIV-damaged capillary endothelial cells in the retina also may facilitate the entry of infected monocytes is to the retina. Infrequently, contiguous spread from the optic nerve may also occur.

CMV retinitis is a full-thickness retinal infection that originates peripherally as perivascular, opaque white granular areas of necrosis associated with hemorrhages. Infiltrates are composed of retinal edema, infected retinal cells, and cellular necrosis affecting all layers of the retina. Vitritis is typically minimal, although reconstitution of the immune system with HAART can lead to more vigorous vitritis. Intranuclear and intracytoplasmic DNA-positive viral inclusions are found within necrotic cytomegalic cells. Untreated, CMV retinitis progresses at a median rate of 24 m/d. Of those patients with CMV retinitis, 35% may present with bilateral disease, and up to 52% will eventually develop bilateral disease.

Contiguous spread is more common than skip lesions. Active virus is present in the advancing edge, with a necrotic retina remaining in its wake. In

clinical studies, progression has been defined as the movement of disease by 750 mm along a 750 mm-wide front or as the development of new CMV lesions. Retinal damage leads to a corresponding absolute scotoma. The thin atrophic retina is susceptible to breaks and rhegmatogenous retinal detachments can occur within 3-6 months of diagnosis. The median time to retinal detachment is 18.2 months. Before HAART, retinal detachments occurred in one third of affected eyes. The larger the area of involvement, the greater the risk of retinal detachment. Retinitis involving 25-50% of the retina conferred a risk 5-6 times greater of retinal detachment than that observed with only 10% involvement. Visual loss is primarily due to retinal necrosis and retinal detachment. Macular edema, CMV papillitis, and neuroretinitis also contribute to visual loss.

Several clinical forms exist. In the fulminant form, diffuse retinal hemorrhage with a whitened necrotic retina similar to a retinal vein occlusion is seen. Patients are often aware of vision loss. The indolent form presents with granular lesions in the peripheral retina often with little or no associated hemorrhage. Patients may notice floaters, or they may be asymptomatic. The third uncommon presentation is frosted branch angiitis. Routine screening with dilated eye examinations has been recommended at 3-month intervals in

patients with CD4+ counts of less than 50 cells/μL because 15% of those patients with active CMV retinitis are asymptomatic.

Differential diagnosis includes HIV retinopathy, toxoplasmosis, *Candida*, syphilis, herpes simplex retinitis, herpes zoster retinitis, and progressive outer retinal necrosis (PORN). Diagnosis is based on the history and the clinical appearance of a white, necrotic, enlarging retinitis, with or without hemorrhage, in a patient who is immunocompromised.

ACUTE RETINAL NECROSIS[4]

- Caused by many viruses especially VARICELLA ZOSTER
- Triad of retinal Necrosis, severe vitritis and vasculitis
- Disease starts as peripheral tongue shaped necrotic lesion and gradually invades the centre.

Progressive outer retinal necrosis

PORN, considered by some to be a variant of ARN, is another common retinopathy, occurring in 2% of patients with AIDS with CD4+ counts of usually less than 50 cells/μL. Peripheral outer retinal necrosis sparing the perivascular retina occurs in a circumferential pattern. These lesions coalesce in weeks and progress to full-thickness retinal necrosis. Posterior progression

occurs with minimal inflammation. Visual deterioration to no light perception occurs within weeks. The fellow eye can be involved in weeks to months.

Differential diagnoses include CMV and toxoplasmosis retinitis. Neither spreads so quickly in a circumferential pattern nor spares the perivascular retina. A clinical diagnosis is made with the appropriate history and the characteristic fundus findings. Diagnosis can be confirmed via PCR on aqueous humor samples or histopathologic and/or immunohistochemical stains on retinal biopsy specimens obtained from the patient.

No effective therapy is available. Intravenous ganciclovir and/or foscarnet given simultaneously with acyclovir may stabilize the infection and delay progression. Intravitreal ganciclovir and foscarnet also have been used with limited success.

RETINAL VASCULITIS AND AUTOIMMUNE DISEASE

BEHCETS DISEASE[54]

Behcet's disease is a multisystem chronic inflammatory disorder, presenting classically with recurrent orogenital ulcers and ocular and skin manifestations. Hypopyon uveitis is the commonest presentation.

Retinal vasculitis major cause of visual morbidity, relapses are frequent, leading finally to retinal and optic dystrophy, variable chorio retinal and retinal pigment epithelial changes, sheathed vessel, chronic mild vitritis.

Yellow white retinal infiltrate are specific not in other idiopathic retinal vasculitis. Recurrent BRVO is common.

Loss of vision is usually after 3 years onset of disease.

HLA- B27 associated [19,56]

Acute Fibrinous anterior uveitis is the most common ocular manifestation of HLA-B27- associated uveitis. If the inflammation is not controlled quickly and adequately, it may result in retinal vasculitis with multiple diffuse foci of retinal infarcts, intraretinal hemorrhages, chronic vitritis, disc swelling, and macular, edema. Retinal vasculitis may not be

clinically apparent, but FFA may disclose peripheral venular vein occlusion with secondary disc neovascularization and marked visual loss can occur.

Human leukocyte antigen is frequently present in patients with ankylosing spondylitis. Reiter's syndrome and psoriatic arthropathy. The retinal vasculitis often responds to periocular steroid injections or high doses of systemic steroids.

BURGERS DISEASE

Arterial wall infiltrate with polymorphonuclear leukocyte, and lymphocyte and inflammation extends into surrounding tissue involving the veins. Fibrosis and proliferation of intima full and lumen obliterated with thrombosis. Periartiritis of retinal vessels results in obliterate end arteritis with thrombosis vessels eventually become sheathed appear as white strands.

SARCOIDOSIS[53,58]

- 27-30 % Ocular involvement
- Idiopathic Granulomatosis disorder of protein manifestation
- Periphlebitis as CANDLE WAX DRIPPINGS – not pathogonomic
- Focal and segment involvement of small vein near equator, hemorrhages, sub retinal infiltrates with RPE atrophy is common.

Treatment

Systemic Corticosteroid resolves with residual sheathing, responds to pan retinal laser photo coagulation.

SYSTEMIC LUPUS ERYTHAMOTSUS

Retinopathy most ocular complication of SLE as mild small vessel disease manifest as multiple cotton wool spots and intra retinal hemorrhage. CWS is hallmark of SLE in contrast DM, HT arteriolar dilation rather than constriction.

Vessel occlusion is caused by true vasculitis with perivascular inflammatory sheathing. This is not true vasculitis, caused by non vascular occlusion, not associated with an inflammatory cell infiltrate and is seen

clinically as CWS. Micro vascular occlusion immune complex, these may penertrate vessel wall as in choroids than an intense inflammatory reaction occur. Large vessel occlusion uncertain, but they may be initiated by lupus anti coagulant, anti cardiolipin antibodies are antiendothelial anti bodies.

Regression induced by pan retinal scatter laser photo coagulation.

WHIPPLES DISEASE

Is a multi system inflammatory disorder that mainly affects men in 5^{th} decade. Intra ocular involvement rare but uveitis, retinal vasculitis and retinal hemorrhage, capillary non perfusion vitreous hemorrhage, and papilledema can occur.

RETINAL VASCULITIS SECONDARY TO MALIGNANCY

CANCER ASSOCIATED RETINOPATHY

Uncommon para neoplastic condition that develops in patient with neoplasia remote from the eye, typically small cell lung cancer patient present with visual loss and progressive night blindness and examination reveals attenuated Retinal vessel.

Retinal phlebitis seen in small cell lung cancer with perivascular sheathing, staining of vessel wall in FFA, no inflammatory cells were identified. It is an auto immune condition with circulating antibodies to retinal cells.

OCULAR LYMPHOMA

Presents as chronic uveitis, manifest as subretinal plaque, retinal infiltrate, hemorrhages retinal vasculitis and retinal pigment epithelial infiltrates.

Tumor cells are clustered in perivascular region of retina, there is destruction of pigment epithelium and bruchs membrane.

Poorly responds to steroid.

DIFFERENTIAL DIAGNOSIS

Based on Age Group[52]

4yrs	Toxo plasmosis, Masquarade syndrome
5-15yrs	Toxo Plasmosis, sympathetic opthalmia, CMV retinititis, leptospirosis
16-40 Yrs	Leptospirosis, Sarcoidosis, Behcets, VKH syndrome, sympathetic ophthalmia, candida.
40 Yrs	Leptospirosis, CMV retinits, Acute Retinal Necrosis, progressive outer retinal necrosis, tuberculosis, candida.
AIDS (Any Age group)	Cytomegalo virus retinitis, Acute Retinal Necrosis, progressive outer retinal necrosis, tuberculosis, candida

INVESTIGATION

Key to diagnose retinal vasculitis as in all medical condition lies in accurate and relevant history numerous studies showed that little additional information is gained by blind investigation of patients and pursuing this is neither time nor cost effective. Investigation specific to eye like Fundus fluorescein angiography is most useful to point the underlying pathology.

1. Slit lamp examination and indirect Ophthalmoscopy
2. Fundus fluorescein angiography –
    - Most useful to point to the underline diagnosis
    - Used in assessing the
        (1) Integrity and density of retinal pigment epithelium
        (2) Permeability and perfusion of retinal vessels
        (3) Presence of neovascularisation
        (4) Extent of disc swelling
        (5) Helpful in management

FFA .5ml 10% Na fluorescein intravenous

After 10-15 seconds faint patchy regions in choroids

With 3-5 minutes dye equally distributed throughout the body

With in 1hour whole dye eliminated

Excitation wave length 465nm blue light emits 525nm yellow green .

Fluorescein leakage[43]

Arteriolar leakage in the presence of intra ocular inflammation is usually due to viral or protozoal infection although retinal arteries may be involved in systemic vasculitis such as systemic lupus erythematosus or wegeners granulomatous this usually leads to occlusion rather than leakage and intraocular inflammation is not a feature of these diseases. Leakage from retinal capillaries alone may occur in syphilis and whipples disease Leakage from post capillary and larger venules. However the most common pattern of retinal vasculitis such as leakage may be focal as seen in sarcoidosis or multiple sclerosis or more diffuse as seen in idiopathic retinal vasculitis, multiple sclerosis and B 27 associated posterior segment.

Macular edema

This is major cause of visual loss in posterior uveitis and potentially reversible and characteristically there is dilation of perifoveal capillary arcade early in FFA with accumulation of dye in cystic space with in retina later on. The degree of associated retinal thickening detected either clinically or by FFA is a good predictor of Visual acuity. Leakage of dye from optic nerve head arises from dilated capillaries and may be either to the primary infiltration or secondary vascular changes induced by intraocular inflammation.

Capillary Closure

Three pattern of retinal ischemia may be identified by FFA. Peripheral capillary closure is feature of tuberculosis, sarcoidosis, and idiopathic retinal vasculitis. Ischemic branch retinal vein occlusions are characteristic of Behcet syndrome and have also been reported in sarcoidosis. Focal capillary dropout at the fovea is often missed because of either media opacity or a failure to identify the fovea at appropriate phase of angiogram. This is an important sign because in presence of unexplained poor visual out come despite adequate suppression of disease. The ability to identify retinal vasculitis an ischemic by FFA has important implication for management.

Neovascularization

Both neovascularisation disc and neovascularisation elsewhere, choroidal new vessel leak flourescein profusely in late phase of FFA. Neovascularisation may occur secondary to wide spread capillary closer are as a direct consequence of intraocular inflammation. It is important to identify the presence or absence retinal ischemia in this situation because management is different. In former case photocoagulation may be indicated as in later immuno suppression will usually induced regression of neovascularisation response.

SPECIFIC INVESTIGATIONS[42]

Suspected Disease	Investigation
Idiopathic Retinal vasculitits	FFA
Sarcoidoisis	Full blood count, GALLIUM SCAN
	Liver function test, CT, CHEST
	X-ray, S.ACE Biopsy
Behcets disease	Skin test
Multiple sclerosis	MRI, CSF, Analysis, VEP
Systemic Vasculitis	ANA, Anti Neutrophil cytoplasmic antibody,
	Erythrocyte Sedimentation Rate, C- reactive
	protein
Tuber culosis	Polymerase Chain reaction from aqueous and
	vitreous samples biopsy
Leptospirosis	Polymerase chain reaction, Micro
	Agglutination titre Toxo plasnosis
	Serology ELISA, DYE test
Syphillis	FTA- ABS test, TPHA test
Viruses	Respective serological titres and Polymerase
	Chain reaction
Malignancy	Full blood count, biopsy CXR, CT scan,
	ocular biopsy for Histopathology

MANAGEMENT

Before embarking on any form of therapy certain point need to addressed they are

- ★ Every efforts made to arrive at diagnosis, especially when dealing with infection and malignant lesion
- ★ Consideration given off treatment needed, as Eale's and par's planitis resolved on its own
- ★ Consider the duration, Risk – Benefit ratio of therapy
- ★ Rule out any irreversible cause of visual loss

Corticosteroids

It remains the main stay of treatment and may be administered topically, regionally or systemically usually by mouth, but also by Intra venous pulse therapy.

Oral prednisolone: 80mg for 4 days than

Follows 60mg for next 14 days

40mg- one month and taper there after according to clinical response.

Periocular steroid:

Effects apparently with in 3weeks and lost for 3months

Triamnicolone di acetate (25mg/ml)

Hydrocortisone acetate (25mg/ml)

Posterior subtenon injection most preferred

Frequent IOP monitor is essential

Systemic immuno suppressive

It is indicated when:

- ★ No response to maximum steroid therapy
- ★ Bilateral disease
- ★ When Visual Acuity decrease by 6/12

Drugs	Dosages
Methotraxate	7.5 – 15mg/week.
Cyclosporin A starting dose	2.5 5mg /kg/day od.
FK506	0.10 – 0.15 mg/kg/day
Chlorambucil	0.1 – 0.2mg/kg/day
Azathioprine	50 – 150 Mg/day
Cyclophosphamide	50-150 mg/day

LASER IN RETINAL VASCULITIS[58]

Mainly indicated in persistent neovascularization causing vitreous hemorrhage.

In India laser used for eales disease

Burns are placed in retinal vascularisation, capillary non perfusion micro aneurysm, and AV shunt vessels.

Direct treatment of laser spot of moderate Intensity (200-500 micron diameter). Elevated new vessels are treated by coagulation of feeder vessel.

VITRECTOMY[42]

In recurrent vitreous hemorrhage, vitrectomy is indicated. The objective is to remove the vitreous hemorrhage in posterior phase, other surgical procedure Lensectomy, Epiretinal Membrane removal, Endolaser and Cryotherapy with Scleral Buckling.

Recommended Treatment Protocol[26]

Patient with idiopathic retinal vasculitis do not necessarily require treatment those with sight threatening complications are started on regime designed to minimize total dose of steroids.

1) Acute Stage: Systemic steroids are started as 0.5 – 1 mg/kg/day. Occasionally intra venous pulse methyl prednisolone 1gm/day for 3 days.
2) Long term control: To allow steroid dose to be reduced, low dose cyclosporine therapy 5mg/kg/day is given. After 3-6 weeks when control has been achieved dose of systemic steroid is tapered and

discontinued. Monotherapy with cyclosporine or combination therapy with low dose steroid can be continued for months/ yrs if necessary.

3) Additional Immunosuppression: Other drugs are required if inflammation persist despite cyclosporine and low dose steroid a variety of agents can be used in combination or as alternative to the maintenance regime. In certain special instances of patients with specific diagnosis such as Wegener's will require additional immuno suppression to control life threatening complication of systemic disease.

Patient who are not responding to therapy also receive the attention of specialists to exclude rare inflammation or malignancy. This is may require unusual investigation that are not routinely available.

REVIEW OF LITERATURE

George et al primary retinal vasculitis; Ophthalmology, Volume 103, Number 3, March 1996 evaluated 25 patients of primary retinal vasculitis. 525 were male and 48% female. The mean age of patients at baseline was 34 years. All patients were bilateral at presentation. The most common finding was vascular sheathing (64%) followed by retinal neovascularisation (40%) sclerotic or attenuated vessels (32%) intra retinal hemorrhage (36%) vascular occlusion (20%) and vitreous hemorrhage (23.1%) vitreous cells are noted in 80%.

Graham EM. Stanford MR, Sanders MD, et al. A point prevalence study of 150 patients with idiopathic retinal vasculitis: 1. diagnostic value of ophthalmological features. Br. J. Ophthalmol. 1989; 73 714-721. Studied 67 patients of isolated retinal vasculitis and 83 patients of retinal vasculitis associated with systemic inflammatory diseases. Out of 67, 27 patients were male and female 72% in age group between 15-40 yrs. Most prevalent finding were sheathing of peripheral retinal vessels in $2/3^{rd}$ of patients, neovascularisation (16%).

Features of 83 patients with retinal vasculitis and systemic inflammatory disease: 44 patients were made and 39 were female. 61% between age group of

15-40 yrs. Ophthalmological features different according to the nature of systemic inflammatory disease.

In Pakistan Journal ophthalmology, idiopathic retinal vasculitis account 49%, inactive tuberculosis retinal vasculitis 27.9% by Dr.Mohamed Sakh Nemon, Shabrol Jaman Siddiqu, Syed. Apr 2004 (2) 53-56.

Vestn Oftalmol. 2001Jul-aug; 117 (4):36-8 Conservative therapy of isolated retinal vasculitis. Ermakova NA. Improvement of visual acuity was more pronounced in patients treated by steroid pulse therapy than in those treated orally and peribulbar. Reabsorption of retinal perivascular exudate was sooner achieved by steroid pulse therapy than by oral treatment.

Gupta et al tuberculosis retinal vasculitis: Retina the Journal of Retinal and vitreous disease 2001 Volume 21 number 5, page 435-444, Evaluated polymerase chain reaction positive tuberculosis retinal vasculitis. 69.2% male and 30.7% were female mean age group of 20 years bilateral in a 9 cases out of 13 cases. The most consistent finding was the presence of vitritis in all eyes. Followed by snow ball opacities in (89.4%) neovascularisation (57.8%) focal Choroiditis (47.3%) vitreous / preretinal hemorrhage (26.3%) and serous retinal detachment in 15.7% over a median follow up of 12 months all showed resolution of vasculitis with no recurrences.

Indian Journal Tuberculosis 2001, 48, 143. Tubercular Etiology in cases of retinal vasculitis*, S.P.Rai, B.N. Panda, V.S. Gurunath and P.K. Sahoo.

A total of 44 cases of retinal vasculitis admitted in the tertiary care centre of the Armed Forces, between January ’98 and June 2000, were evaluated prospectively for evidence of healed or active tuberculosis in the body. Retinal vasculitis was bilateral in 28 and unilateral in 16 patients, all were males; the average age was 31.7 years (range 16 to 53 years); only 2 patients had constitutional symptoms and no patient had past history of tuberculosis.

Retinal disease in patients with systemic lupus erythematosus, Osamu Ushiyama, Keiko Ushiyama, syuichi Koarada, Yoshifumi Tada, Noriaki Suzuki, Akihide Ohta, Shinji Oono, Kohei Nagasawa. The findings included haemorrhages, vasculitis, cotton wool spots, and hard exudates, all of which were considered to reflect vascular damage.

American Journal Opthalmol 1998: 125:312-324. Ralph D. Levinson MD, Robin Vann MD, Janet V. Davis MD, Dorothy N. Fried Berg MP, PhD., Adnan Tufail MBBS, Frcophth, Briant, Terry MD, Jannete I, Lindley MD FRCS (C) and Gary N. Hollan, MD. HIV patients with complaints floaters and blurred vision seen with significant inflammatory sheathing of peripheral retinal

venules minimal vitritis and typical retinal lesions were gray-white to yellow irregular in shape, located in mid periphery of retinal fundus.

Malinowski et al, Long term visual outcome and complication associated with pars planitis, Ophthalmology 100, 818-825; 1993. Report the complication on 54 patients. Most commonest cause of visual loss is cystoid macular edema (50.9%), followed by cataract (41.4%), neovascularisation (6.5%), severe cellophane retinopathy (6.5%) and retinal detachment (8.3%).

AIM OF THE STUDY

- ★ To determine the various etiological pattern of retinal Vasculitis
- ★ To determine the various ophthalmoscopic features at presentation.
- ★ To assess visual outcome in various etiological retinal Vasculitis

MATERIALS AND METHODS

It was a prospective study of patients who attended the retina and uvea service at Ophthalmology Department Government Rajaji Hospital Madurai.

All cases of Retinal Vasculitis were taken up for study, irrespective of other ocular or systemic manifestation.

A standard proforma was used to collect and document all the details regarding the cases included in the study.

The detailed information regarding history and specific questions about presence of possible multisystem inflammatory disorders were asked.

History of

orogenital ulcerations and skin rash (Behcet's syndrome),

recent weight loss, dry cough, night sweats, arthralgia (sarcoidosis)

neurological symptoms (Multiple sclerosis and sacoidosis),

arthritis (sero negative arthropathies),

and recent changes in bowel habits.

The absence of any diagnostic clues from history makes idiopathic retinal vasculitis most likely.

The diagnostic criterion of retinal vasculitis was made atleast one of the following ophthalmoscope features.

1) Sheathing
2) Perivascular Edema
3) Perivascular inflammation
4) Staining of vessel wall in FFA.

Retinal arteries involvement is almost always due to systemic vasculitis or viral retinitis, capillary involvement occurs in syphilis and whipples. Retinal vein involvement occurs in sarcoidosis, Behcets, Multiple sclerosis, inflammatory bowel disease. Intra retinal infiltrates are characteristics of infectious process. Cotton wool spots in systemic vasculitis. Swelling of optic nerve head is a common non specific finding related to intra ocular inflammation.

After collecting detailed information regarding history and complaints, a thorough examination of anterior segment was done with slit lamp. Intraocular pressure recorded with schiotz tonometer. Gonioscopy was done routinely for all cases. Retinoscopy was done with or without clear media to assess refractive status.

For examination of posterior segment all the cases were subjected to dilated ophthalmoscopic evaluation with fundus lens and indirect

ophthalmoloscopy. Fundus Flourescien Angiography was done in appropriate cases.

All cases were subjected to following lab Investigations

1. Haemogram – Total count, Differential count, Erythrocyte sedimentation rate.
2. Urine albumin sugar
3. Mantoux
4. Blood VDRL
5. Blood sugar
6. Bleeding time / clotting time

All the cases were referred to the following departments attached to our Govt. Rajaji Hospital Madurai

1. Rheumatology clinic
2. Ear nose throat clinic
3. Dermatology Department
4. Chest clinic
5. Sexually Transmitted Disease department
6. Neuro medicine Department
7. Dental Department

Diagnosis was based mainly on history, ophthalmoscopic findings at presentation; clinical features are correlated with the results of various investigations at presentation.

In the absence of systemic history and inconclusive laboratory investigation, ophthalmoscopic finding suggesting primary involvement of retinal vessels with significant vitritis are grouped as idiopathic or primary retinal vasculitis.

PRIMARY RETINAL VASCULITIS

1. For cases with peripheral Vasculitis and good visual Acuity with no Evidence of retinal non-perfusion, periodic observation was made.

2. For cases with peripheral vasculitis and good visual Acuity with evidence of less than 5 DD retinal non perfusion, systemic steroids, prednisolone 60mg/day in divided dose for a period of 2 weeks were given then tapered. Then periodic review was advised.

3. For cases with peripheral Vasculitis associated with a relative non perfusion of more than 5 DD, retinal photocoagulation was advised.

4. For cases of vitreous hemorrhages with neo vascularization and some amount of clear media laser photocoagulation was advised.

5. For cases of vitreous haemorrhage were advised observation and regular follow up. In cases of Vitreous haemorrhage with opaque media and evidence of tractional bands in Ultrasonography transconjunctival cryopexy was advised. If vitreous hemorrhage did not resolved with in 6 months vitrectomy was opted.

6. For cases of tractional Retinal detachment vitrectomy with Endophotocoagulation was advised

SECONDARY RETINAL VASCULITIS

For cases of Vasculitis secondary to ocular and systemic disorders management was directed towards associated condition.

RESULTS AND OBSERVATION

This prospective study of 41 patients Retinal Vasculitis were analysed as follows

A. Distribution of etiology

In this study of 41 cases

Diseases	Cases	Percentage
Retinal Vasculitis of known etiology	**22**	**53.65**
Tuberculosis	10	24.39
Human Immuno deficiency Virus	7	17.07
Systemic lupus erythematosus	4	9.75
Pars Planitis	1	2.43
Idiopathic Retinal Vasculitis	**19**	**46.34**
Total	41	100

In this study retinal vasculitis of known etiology accounts for 53.65% (22 cases) and unknown etiology retinal vasculitis of 46.34% (19 cases).

Commonest cause of retinal vasculitis in this study was associated with idiopathic retinal vasculitis 19 cases (46.34%). Other major causes are Tuberculosis (24.39%), Systemic lupus erythematosus 4 cases (9.75%) 1 (2.43%) case of Intermediate uveitis and 7 (17.7%) cases of Human Immuno deficiency Virus positive.

B. Sex Incidence of retinal vasculitis

Sex	Idiopathic Retinal Vasculitis	Tuber culosis	Systemic lupus erythematosus	Human Immuno deficiency virus positive	Pars planitis	Percentage
Male	11	7	1	4	1	58.53
Female	8	3	3	3	-	41.46

In this study a slight male preponderance (58.53%) is in compare to female 41.46% Murphy et al noted equal Involvement among men and women. Idiopathic retinal vasculitis, tuberculosis has a significant male proportion, where as systemic lupus erythematosus predominant in female.

C. Age Distributions

Age group	Idiopathic Retinal Vasculitis	Tuber culosis	Systemic lupus erythematosus	Human immuno deficiency virus	Pars planitis
<20	4	-	-	-	-
21 – 30	8	2	2	5	-
31-40	5	4	2	2	1
>40 yrs	2	4	-	-	-

Most cases of retinal vasculitis occurred between second and third decade of life. In this age group our study of idiopathic retinal vasculitis around (68.4%) and tuberculosis (60%).

D. Laterality

	Cases	**Percentage**
Unilateral	18	43.90
Bilateral	23	56.09

Among 41 cases of RV 23 cases (56.09%) were bilateral. Most cases of retinal vasculitis are initially presents unilateral, over a period of time it developed into bilateral.

E. Mode of presentation

Onset	**Idiopathic Retinal Vasculitis**	**Tuber culosis**	**Systemic lupus erythematosus**	**Human immuno deficiency virus**	**Pars planitis**
Acute	10	7	0	2	-
Chronic	9	3	3	3	1

Acute onset is 19 (46.34%) cases chronic onset around 19 (46.34%) cases idiopathic retinal vasculitis has equal proportion of onset. Where as tuberculosis and virus related retinal vasculitis are acute onset. 3 cases were asymptomatic.

SYMPTOMS

Symptoms of 64 eyes studied as follows

Symptoms	Idiopathic Retinal Vasculitis	Tuber culosis	Systemic lupus erythematosus	Human immuno deficiency virus	Pars planitis
Defective vision	12	6	3	2	-
Floaters	2	2	1	4	1
Redness	1	3	-	-	-
Pain	1	1	-	2	-
Photophobia	-	-	-	0	1
A symptomatic	-	-	1	2	-
All of the above	3	1	-	-	-

Defective vision is the commonest complaint of patients account for 35.9%. HIV patient has complaints of floaters mainly. One case of systemic lupus erythematosus and two cases from antiretroviral center referred for routine fundus examination were asymptomatic. Pans planitis has complaint of photophobia and floaters.

POSTERIOR SEGMENT FINDINGS AT PRESENTATION

Symptoms	Idiopathic Retinal Vasculitis	Tuber culosis	Systemic lupus erythematosus	Human immuno deficiency virus	Pars planitis
Vascular sheathing	14	8	3	3	1
Neo vascularisation	8	3	-	-	-
Intra retinal Haemorrhage	6	2	3	0	-
Sclerotic vessel	8	2	2	-	-
Cystoid macular edema	3	3	-	1	-
Vitreous Haemorrhage	6	1	-	-	-
Vascular occlusion	-	-	1	-	-
Retinal Detachment	2	-	-	-	-
Vitritis	6	5	2	2	1
Disc edema	-	-	-	2	-

In idiopathic retinal vasculitis posterior segment finding of vascular sheathing seen in 14 eyes (46.6%), neovascularisation 8 eyes (26.6%), vitreous hemorrhage 6 eyes (20%).

In tuberculosis retinal vasculitis sheathing 50% of eyes are involved. In systemic lupus erythematosus patient shows cotton wool spot, hard exudate, 3 eyes of perivascular sheaths and 3 eyes with intra retinal hemorrhage.

In pars planitis, peripheral vascular sheathing is present and with significant vitritis.

TREATMENT

In addition to specific treatment, corticosteroid was main stay of therapy in almost all cases of retinal vasculitis irrespective of etiology. They were used by pulse therapy as I.V methyl prednisolone sodium succinate 1g daily for 3 days then Prednisolone 1 mg/kg/day doses is given and then tapered gradually. Pulse therapy is not used in tuberculosis and human Immuno deficiency virus positive patients. Two cases of recurrent vitreous Hemorrhage referred to Vitreo retinal centre.

One case of Tract ional bands and neovascularisation were advised for laser photocoagulation Human Immuno Virus positive and Systemic Lupus Erythematosus referred to there respective clinics for further management which improves the visual acuity much better. In one case of pars planitis with retinal vasculitis regular follow up, no treatment given.

STATUS OF VISUAL ACUITY IN 64 EYES AFTER 6 MONTHS

Disease	After 6 months			Patient not turn over
	Improvement	Static	Deterioration	
Idiopathic retinal vasculitis	6	6	14	4
Tuber culosis	10	6	0	-
HIV	2	1	5	1
SLE	2	3	3	-
Pars planitis	-	1	-	-

Idiopathic retinal vasculitis shows improvement of two lines in 23.07% of eyes, static 23.07%, deterioration 53.8%, tuber culosis retinal vasculitis shows great improvement around 62.50% static about 37.5%. Human Immunodeficiency Virus shows visual deterioration in spite of HAART therapy. Systemic lupus erythematosus 37.5% of eyes shows visual deterioration, which indicates the severity of disease.

DISCUSSION

This study of 41 patients with retinal vasculitis is to assess the underlying etiology, to analyse the course of disease and to evaluate their visual outcome.

Most of published literatures were from West and there etiology most commonly, account for systemic auto immune disorder.

In our study systemic lupus Erythematosus account for 9.75% and no Behcet's disease. On a study of Pakistan Journal ophthalmology Idiopathic retinal vasculitis account 49% inactive tuberculosis retinal vasculitis 27.9% by Dr. Mohamed Sakh Nemon et al[16].

In our study major etiological factor are primary or Idiopathic Retinal Vasculitis accounts for 46% and infectious, both tuberculosis and human immuno deficiency virus accounts for 41.46% other contributing factors are systemic disorders like systemic lupus erythematosus in 9.75% and pars planitis in one case.

Eales disease is also a diagnosis of exclusion and, a form of primary periphlepitis like isolated or primary retinal vasculitis, it becomes hard to separate the two entities But, currently Eales disease is considered to effect

multiple quadrants of the retina with progression of capillary drop out from periphery towards the posterior pole, and more over increased frequency of vestibulo auditory findings are seen in patients of Eales disease. While most of the patients of primary retinal vasculitis have diffused vasculitis and capillary drop out of both peripheral and central retina. However, with out a clear understanding of pathophysiology of two entities, it is certainly possible that they represent a continuation of similar disease process as by George et al[31].

In 19 cases of idiopathic retinal vasculitis 57.8% were male and 42.10% female. Among which 13 cases in the age group of 20-40 years, most of cases were bilateral at presentation Posterior segment finding of idiopathic retinal vasculitis were vascular sheathing in 46.6% (14 eyes), vitreous hemorrhage in 20% (6eyes), vitritis was significant, neovascularization disc, neovascularization elsewhere hallmark of idiopathic retinal vasculitis.

Visual acuity of 6 eyes shows improvement of more than 2 lines from Snellen chart using steroid alone. Using pulse steroid therapy reabsorption of retinal perivascular exudates was quicker than using oral steroid alone. 6 eyes vision remains static and 14 eyes shows vision deterioration.

George et al[31] accounts for 64% of vascular sheathing, neovascularisation 46% vitreous hemorrhage 24%.

Second contributing factor for retinal vasculitis is infections due to tuberculosis and HIV. Tuberculosis retinal vasculitis in 10 cases, most cases shows some radiographic evidence of post pulmonary tuberculosis. 2 cases with mantoux positive, Age group around 20 – 40 yrs, bilateral, male with posterior segment findings shows vascular sheathing 50% moderate vitritis cystoid macular edema, neovascularization patient with anti tuberculosis treatment shows great improvement of visual acuity around 62.5%. In Gupta et al[34] 69.2% male and 30.7% were female mean age group of 20 years bilateral in a 9 cases out of 13 cases. The most consistent finding was the presence of vitritis in all eyes. Followed by snow ball opacities in (89.4%), neovascularisation (57.8%), focal Choroiditis (47.3%), vitreous / preretinal hemorrhage (26.3%) and serous retinal detachment in 15.7% over a median follow up of 12 months all showed resolution of vasculitis with no recurrences.

Incidence of Systemic lupus erythematosus in our study is 9.75% had features of vessel sheathing, narrowing or tortuous. In Ermakova NA et al[15] shows 34.5% retinal vascular abnormality (3%) retinal vasculitis with extensive peripheral capillary non perfusion and neovascularization.

Referred patient as HIV positive with complaints of floaters and Blurred vision seen with significant inflammatory sheathing of peripheral retinal venules minimal vitritis and typical retinal lesions were gray-white to yellow

irregular in shape, located in mid periphery of retinal fundus. 55.5% visual out come is poor inspite of highly active antiretroviral therapy (HAART).

Our study had one case of intermediate uveitis, with feature of peripheral retinal vein are dilated, segmental and tortuous with white sheath and small patches vein wall associated with minimal vitritis. Visual acuity is normal, no treatment is given and patient is on follow up.

Out of 41 cases 5 cases developed complicated cataract all had posterior subcapsular cataract may be due to steroid therapy or due to chronic inflammation.

CONCLUSION

With in the limitations of this study, examination of these 41 patients has shows, different pattern of retinal vasculitis. And it is mandatory for all cases of retinal vasculitis to be examined in detail in order to look for ocular involvement other systemic involvement and to do a complete work up of investigation procedures. This will certainly help to classify the retinal vasculitis.

It is evident that when case of idiopathic retinal vasculitis are detected early and treated adequately the incidence of vision threatening complication are considerable minimized. Vasculitis associated with human Immuno deficiency virus had a worse outcome due to rapid progression despite therapy.

More follow up period is necessary to describe the complications of the disease and also to analyze whether case of Idiopathic Retinal Vasculitis develop any systemic feature would lead on to a specific diagnosis.

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ABBREVIATIONS

AIDS	-	Acquired Immunodeficiency Syndrome
ANA	-	Anti Nuclear Antibody
BRVO	-	Branch Retinal Vein Occlusion
BT	-	Bleeding Time
CMV	-	Cytomegalo Virus
CRVO	-	Central Retinal Vein Occlusion
CSF	-	Cerebro Spinal Fluid
CT Scan	-	Computed tomography
CT	-	Clotting Time
CWS	-	Cotton Wool Spots
FFA	-	Fundus Fluorescence Angiography
Govt	-	Government
HAART	-	Highly Active Antiretroviral Therapy
HIV	-	Human Immunodeficiency virus
LE	-	Left Eye
MRI	-	Magnetic Resonance Imaging
NVD	-	Neovascularisation Disc
NVE	-	Neovascularisation Elsewhere
RE	-	Right Eye
RVH	-	Repeated Vitreous Hemorrhage
SLE	-	Systemic Lupus erythematosus
TB	-	Tuberculosis
VEP	-	Visually Evoked Potential
VKH	-	Vogt Koyanagi – Harada Syndrome
VDRL	-	Veneral Disease Research Laboratory

PROFORMA

I. PATIENTS RECORD

Name Age Sex Male /Female:

Address Outpatient Number

II. COMPLAINT

Laterality RE LE BE

Symptoms: Defective vision Duration

Floaters Onset

Pain Severity

Redness Course

Any Previous attack

Past Ocular History

Intra Ocular Inflammation

Prior Visual Loss - Right eye/ Left eye

Miscellaneous

SYSTEMIC HISTORY

General fever/malaise / loss of weight/loss of appetite

Skin Rashes / subcutaneous nodules / urticaria / edema / Raynaud phenomenon/ alopecia / ulcers

Joints	Arthritis / Arthralgia / Low back pain / Inflammatory signs.	
Pulmonary	Chronic cough	Hemoptysis
Neurological	Headache / Thickened Nerves / Tinnitus.	
Renal	edema of legs / puffiness of face / polyuria / oliguria	

H/O INTAKE OF DURGS

Treatment history	On steroid	1
	Not on steroid	2
	Anti metabolites	3

Ocular Examination :

Laterality	LE	RE	BE
Anterior segment	Granulomatosis	Non-granulomatosis	
Conjunctiva			
Nodules			
Lacrimal grand			
Sclera			
Cornea			
AC Reaction	Hypopyon / hyphema		
Iris			
Intra ocular pressure	RE	LE	

Posterior segment Vitreous Disc

VASCULITIS PATTERN

Arterioles	Segmental Vasculitis
Veins	Diffuse Vasculitis
Both	Occluded Vessel
Capillaries	Sheathing
Retinal infiltrate	Pars planitis
Chorioretinal scar	Macular Edema
Neo vascularization	Retinal Hemorrhage
Choroidal Granuloma	Any Sequence
Vitreous Hemorrhage	

SYSTEMIC FINDING

INVESTIGATION

Blood pressure	VDRL
Urine: albumin, sugar	ELISA
Hemoglobin	Toxo Igm
Total count	Motion Ova/cyst
Differential count	
BT / CT	
ESR	
Mantoux	

Chest X-ray

Fundus flourescein Angiography RE LE

Early – Hypoxic Area New vessels disc and New vessels else where

Late - Hypoxic Area RE LE

TREATMENT

Steroid

Immuno suppressive

Surgery (vitrectomy)

Others

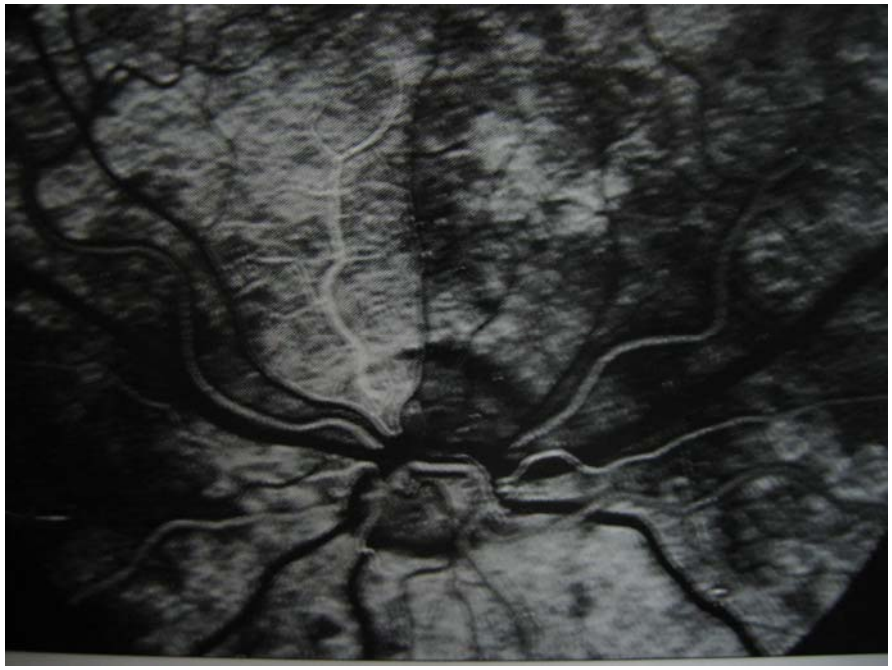
DIAGNOSIS

FOLLOW –UP

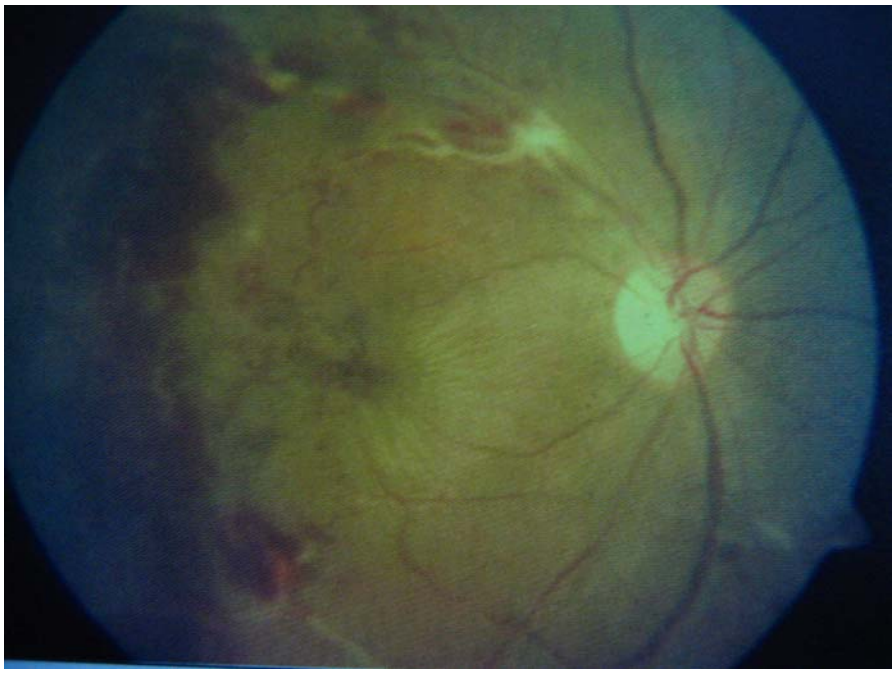
Date	Clinical Features	Remarks

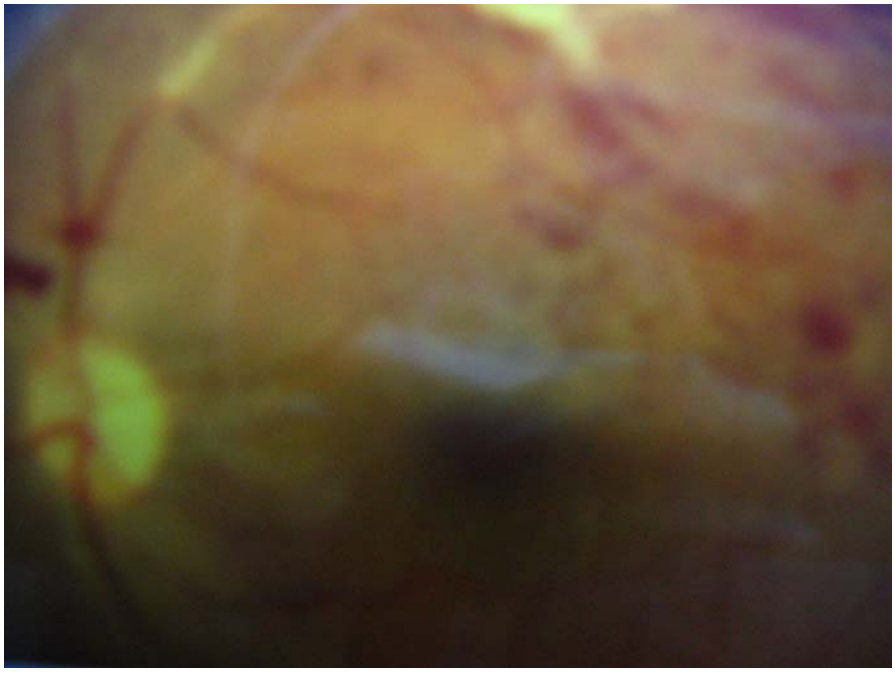
NORMAL FUNDUS

NORMAL FUNDUS FLUORESCEIN ANGIOGRAPHY



IDIOPATHIC RETINAL VASCULITIS

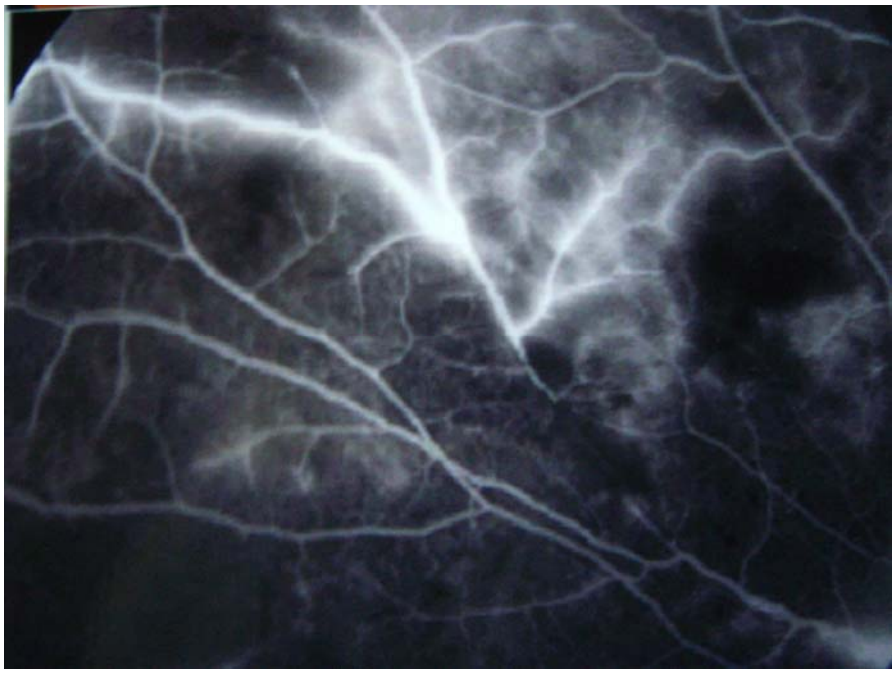




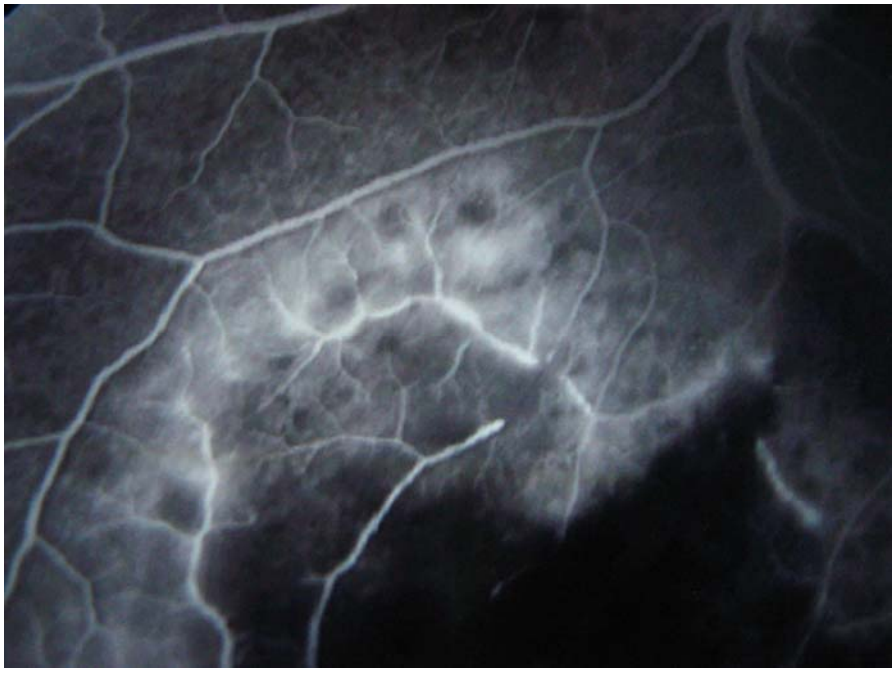
VASCULITIS IN TUBERCULOSIS

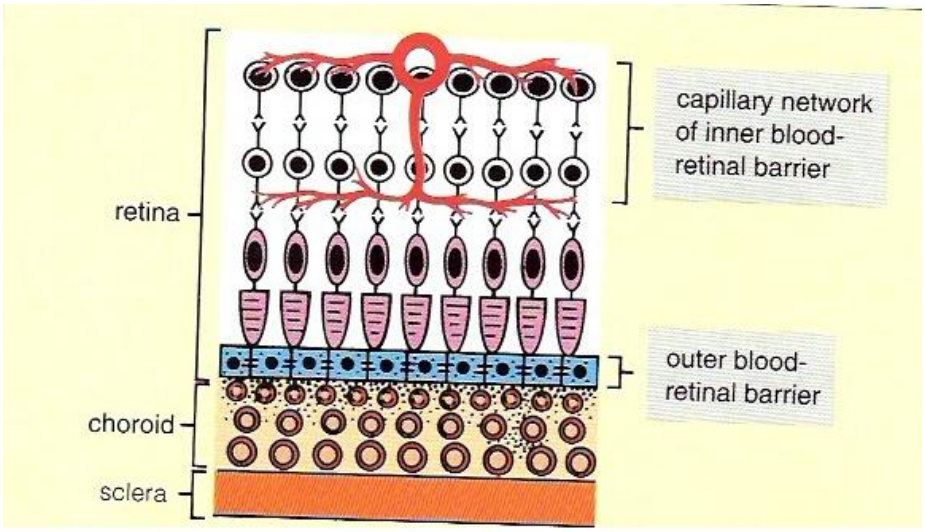
VASCULITIS IN CYTOMEGALO VIRUS

FFA - STAINING OF VESSEL WALL IN RETINAL VASCULITIS

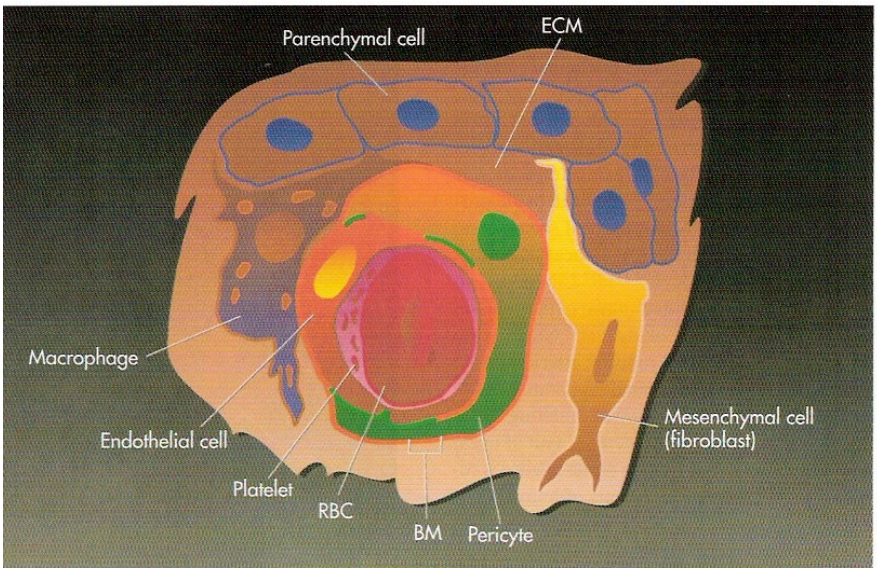


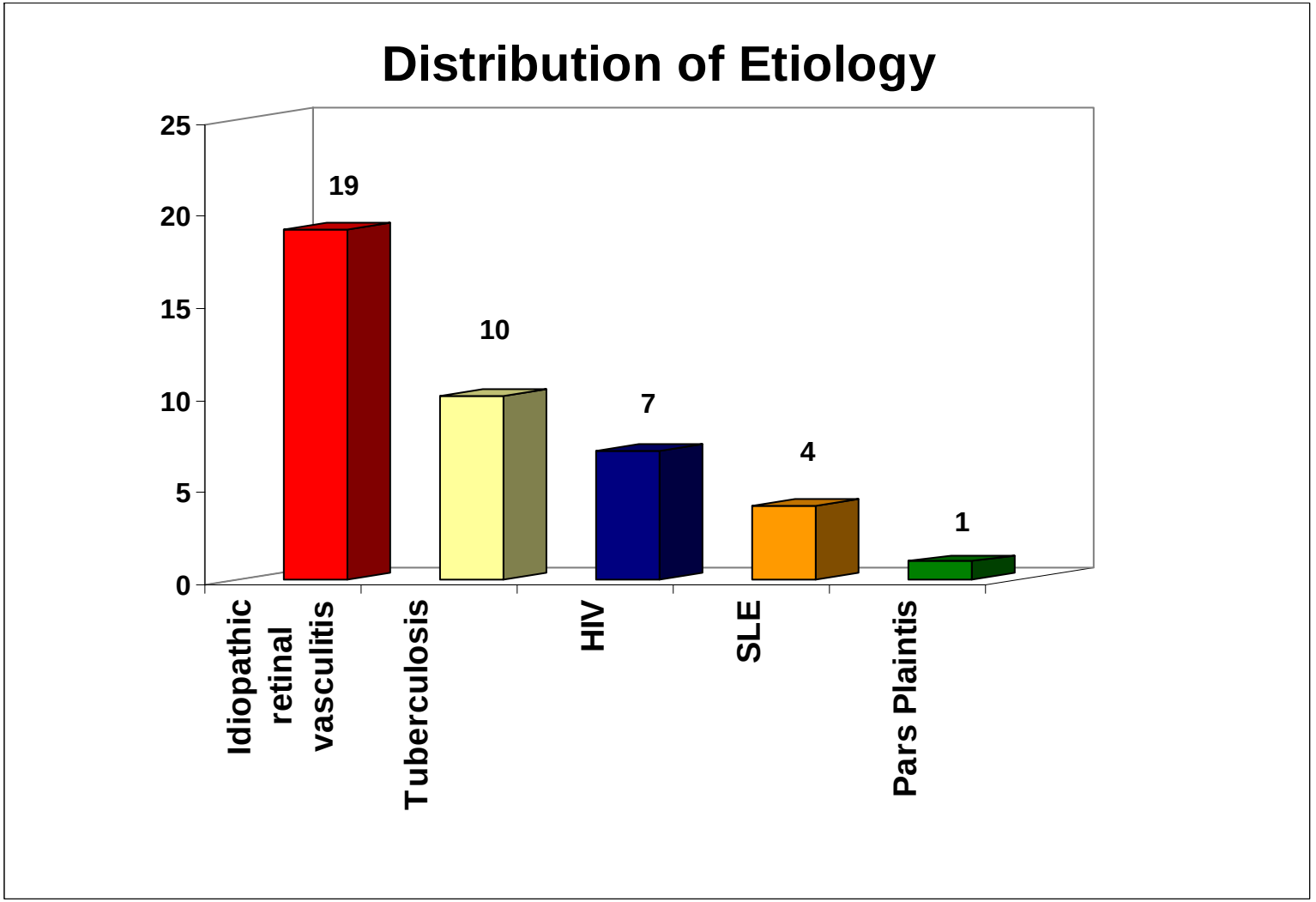
FFA
LEAKAGE DUE TO NEOVASCULARISATION

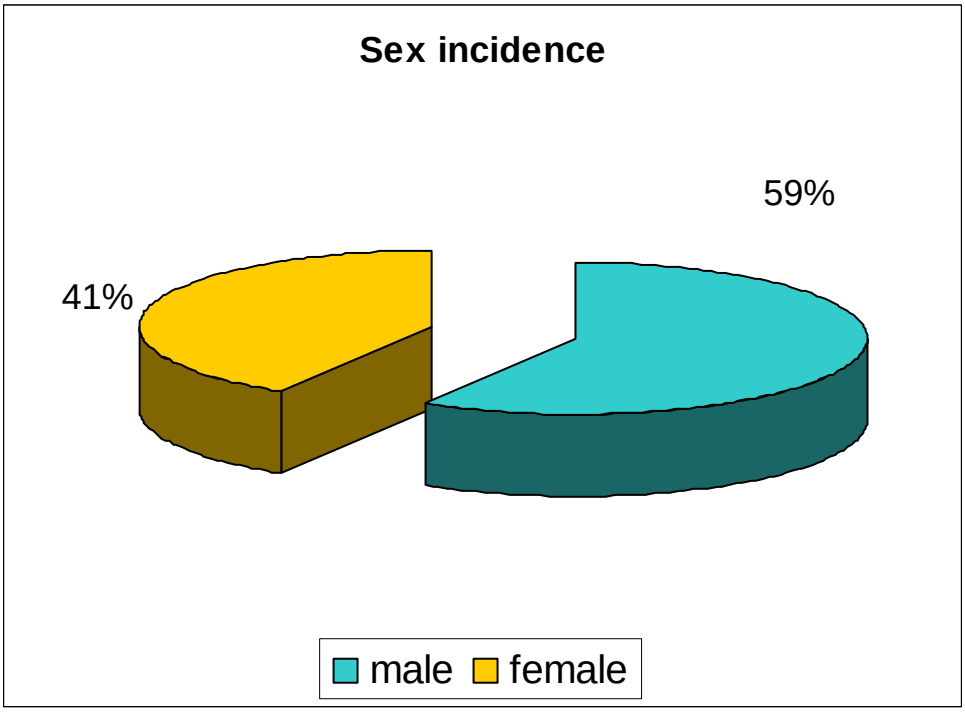


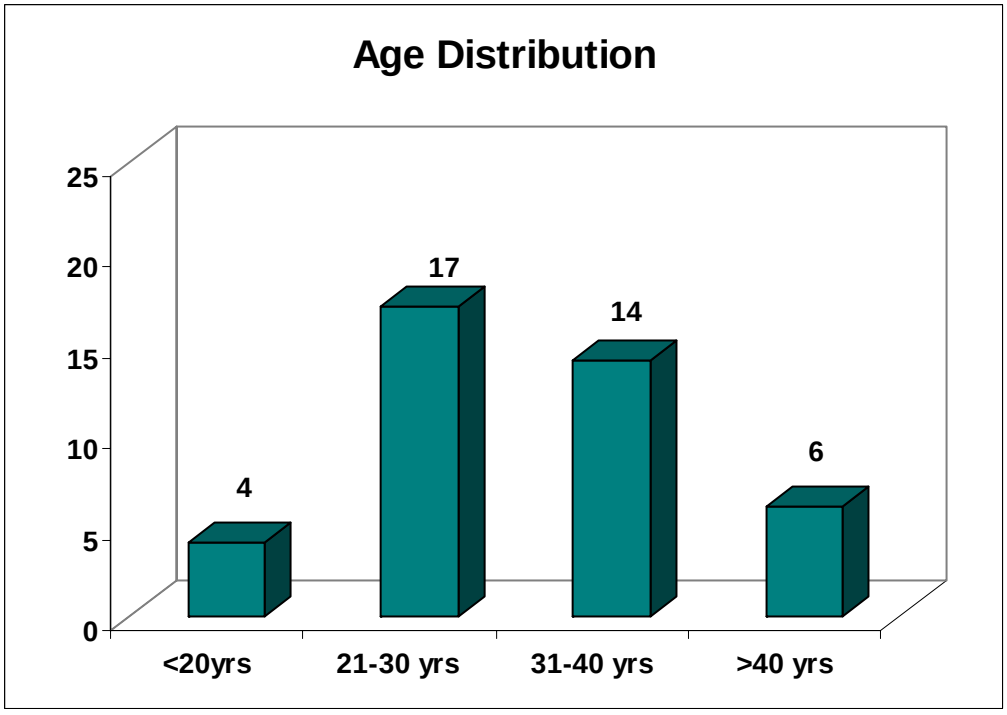


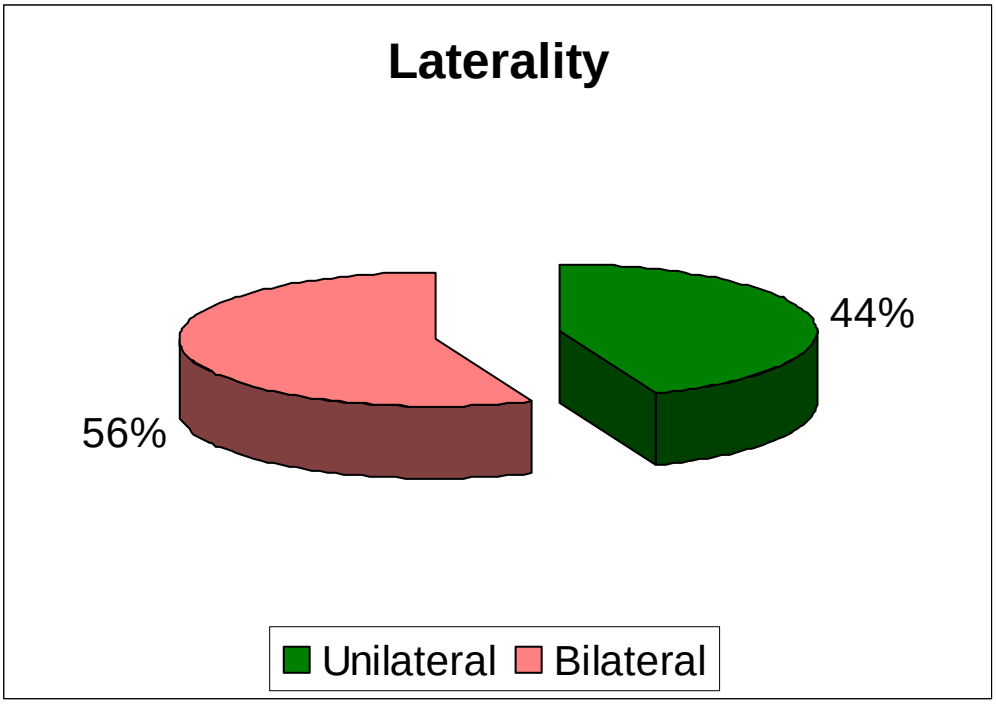
NORMAL RETINAL MICROVASCULAR COMPONENTS

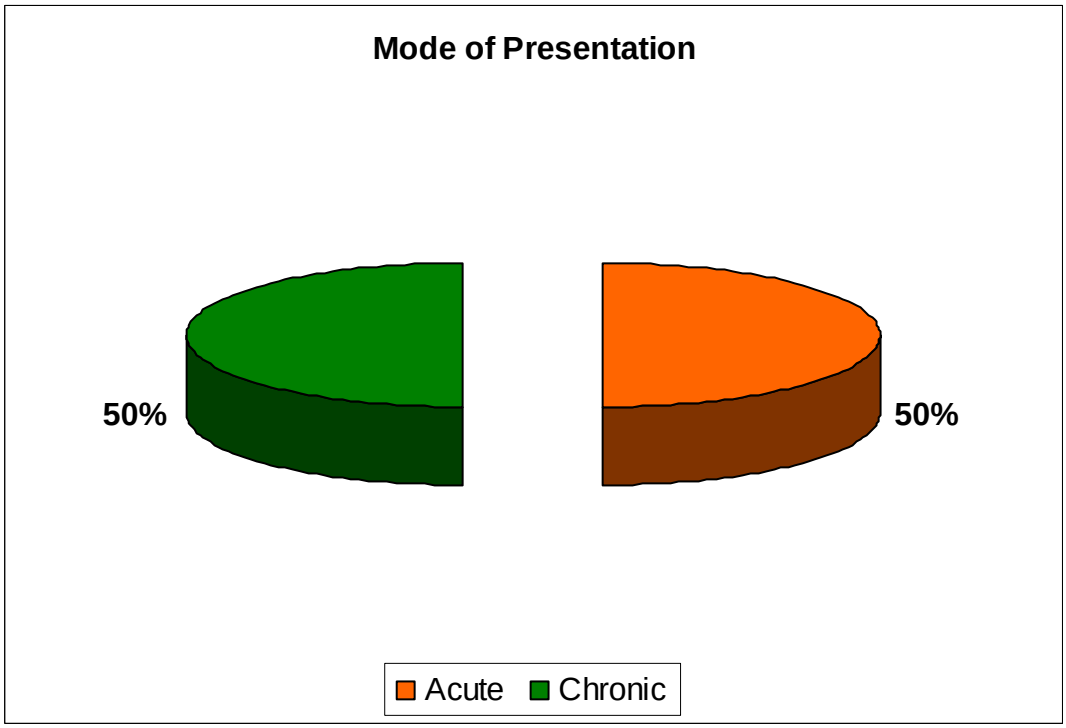


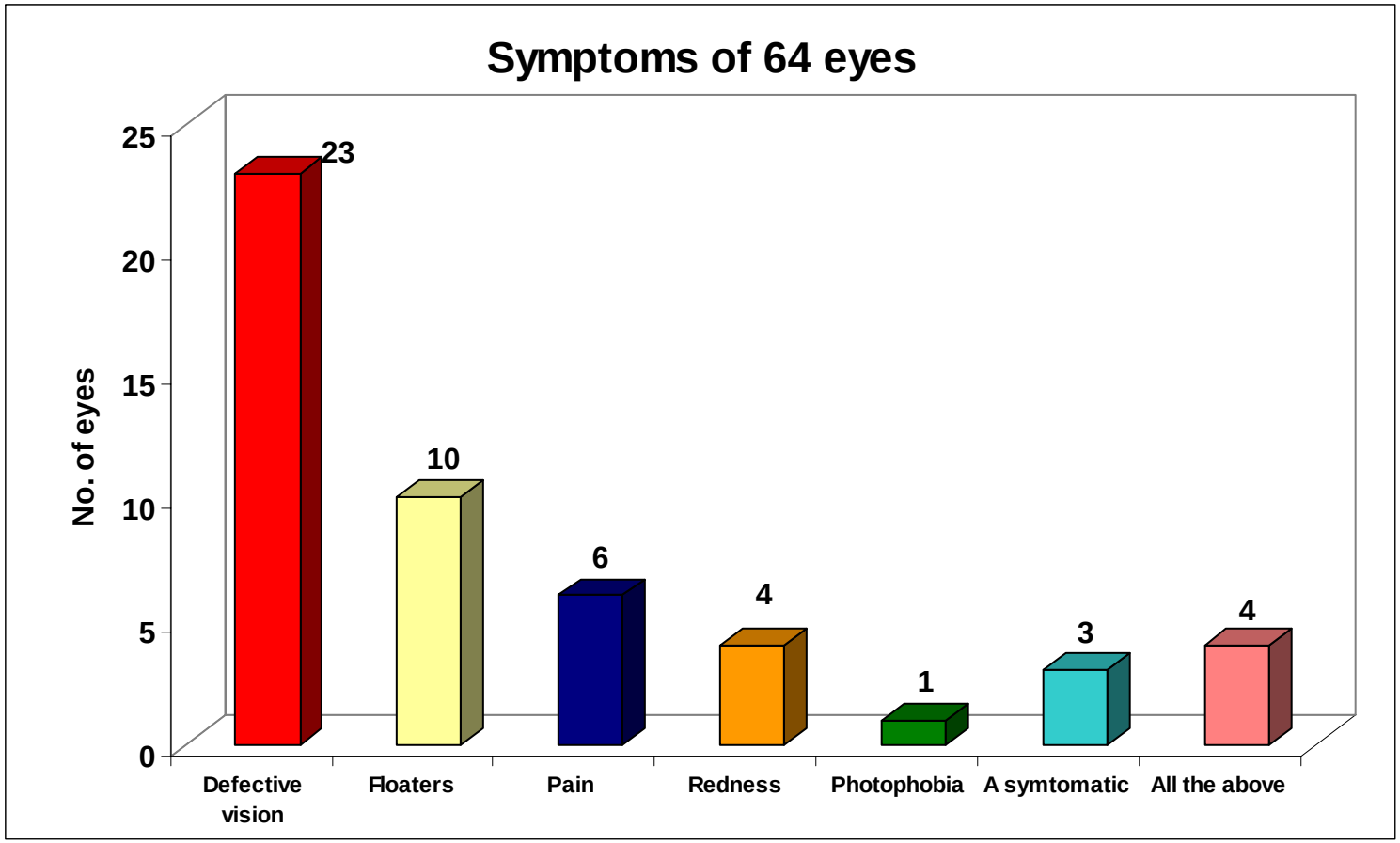

Distribution of Etiology
25
20
15
10
5
0
19
10
7
4
1
Idiopathic retinal vasculitis
Tuberculosis
HIV
SLE
Pars Plaintis

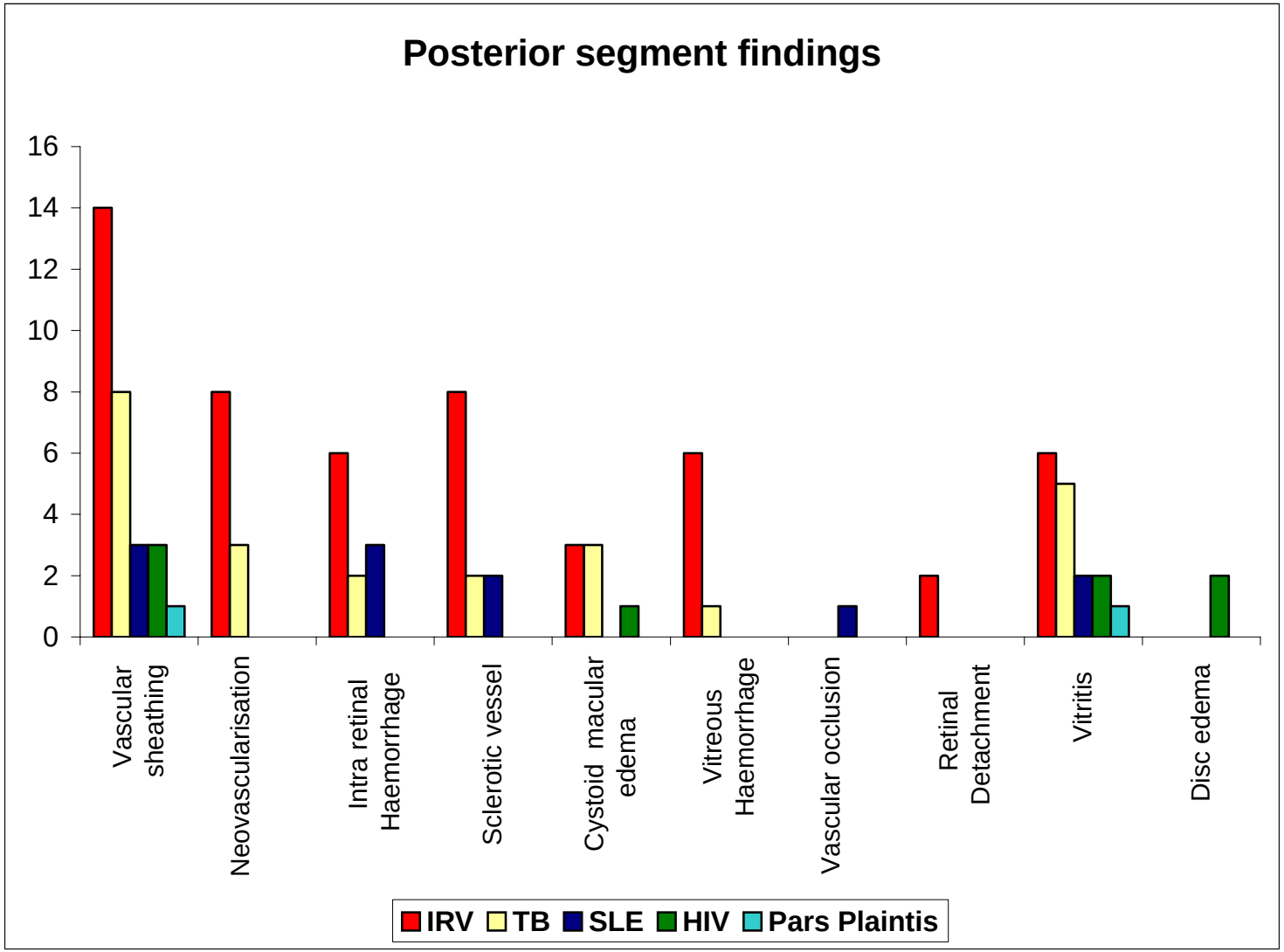

Sex incidence
59%
41%
male
female

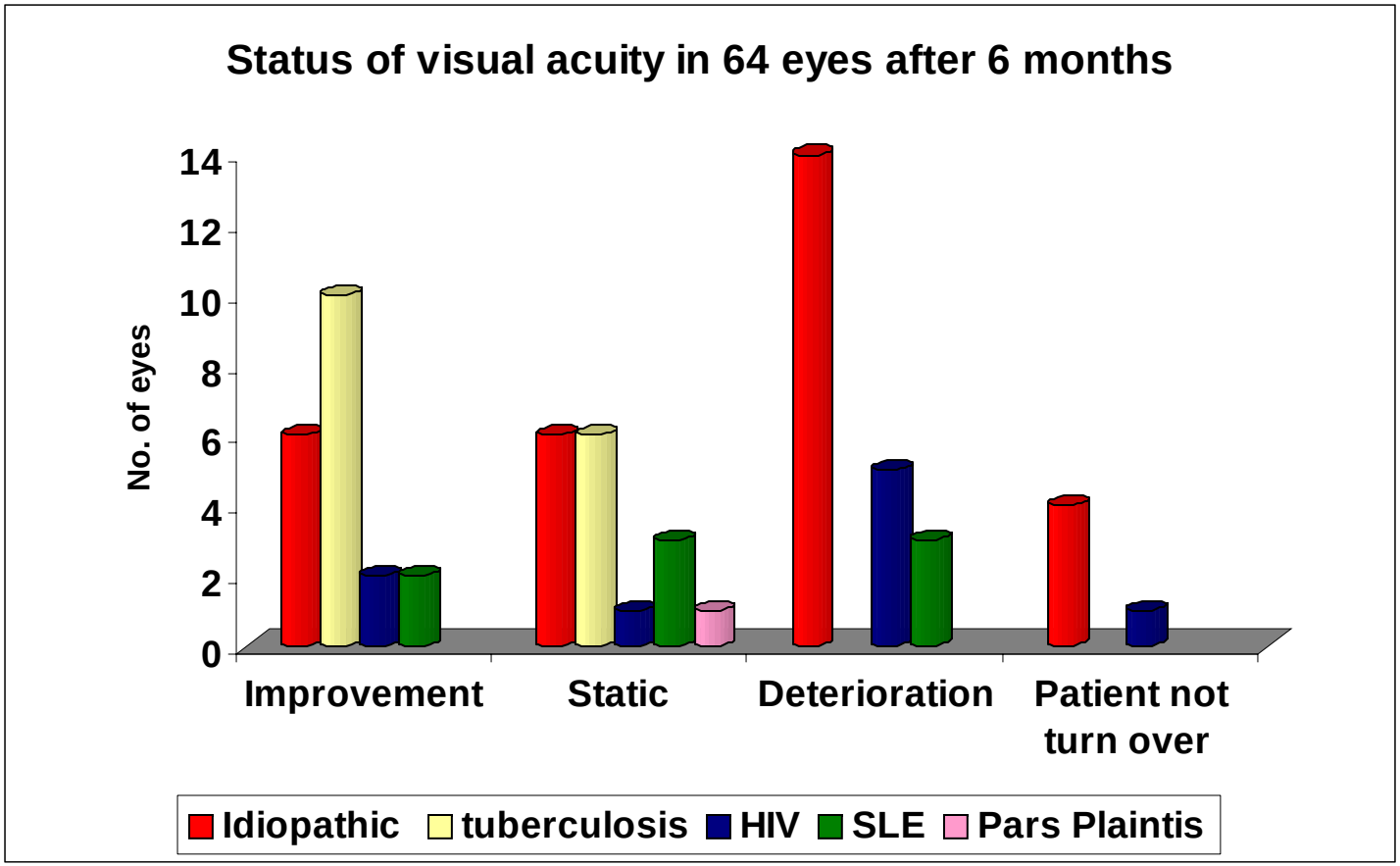

Age Distribution
25
20
15
10
5
0
4
17
14
6
<20yrs
21-30 yrs
31-40 yrs
>40 yrs


Laterality
44%
56%
Unilateral
Bilateral


Mode of Presentation
50%
50%
Acute
Chronic


Symptoms of 64 eyes
No. of eyes
25
20
15
10
5
0
23
10
6
4
1
3
4
Defective vision
Floaters
Pain
Redness
Photophobia
A symtomatic
All the above


Posterior segment findings
16
14
12
10
8
6
4
2
0
Vascular sheathing
Neovascularisation
Intra retinal Haemorrhage
Sclerotic vessel
Cystoid macular edema
Vitreous Haemorrhage
Vascular occlusion
Retinal Detachment
Vitritis
Disc edema
IRV
TB
SLE
HIV
Pars Plaintis


Status of visual acuity in 64 eyes after 6 months
No. of eyes
14
12
10
8
6
4
2
0
Improvement
Static
Deterioration
Patient not turn over
Idiopathic
tuberculosis
HIV
SLE
Pars Plaintis

Master Chart

S.NO	NAME	AGE	SEX	SYMPTOMS	MODE OF ONSET	LATERALITY	SYSTEMIC DISORDER	ASSOCIATED OCULAR DISORDER	VITREOUS HAEMMORHAGE	NEO VASCULARISATION	INITIAL VISION		VISION AFTER 6 MONTHS		TREATMENT & FOLLOW UP
											RE	LE	RE	LE	
1	Palani Samy	22	M	Def.vision	A	BE	N	N	+		6/6	6/36	6/60	HM	Ref. to vitreo retinal centre
2	Karupasasumathy	18	F	Floaters	A	LE	N	N	+		6/6	6/12	6/6	HM	
3	Saravanan	25	M	Pain	A	LE	HIV+	N			6/6	6/12	6/6	6/60	HAART
4	Sairam	44	M	Def.vision	A	BE	TB	N		+	6/12	6/9	6/12	6/9	On RNTCP
5	Kanchana	35	F	Floaters	C	BE	HIV +		-	-	6/18	6/36	6/12	6/12	HAART
6	Sankar	22	M	Def.vision	A	BE	TB	N			6/18	6/18	6/9	6/18	RNTCP
7	Jayanthi	31	F	Asymptomatic	-	BE	SLE	N	-	-	6/6	6/6	6/6	6/6	
8	Rajamahamed	19	M	Photophobia	A	RE	N	N			6/9	6/6	-	-	Patient not turn over
9	Rajamahamed	26	M	Def.vision	C	BE	N				6/12	6/12	6/12	6/12	
10	Muthulakshmi	48	F	Redness	C	LE	TB	N			6/36	6/36	6/9	6/9	RNTCP
11	Manikandan	33	M	Def.vision	A	LE	TB				6/9	6/36	6/9	6/9	RNTCP
12	Mariammal	37	M	Pain, Def.vision	C	LE	N	N			6/6	6/9	6/6	6/24	
13	Murugan	29	M	Def.vision, pain, photophobia	A	BE	N			-	6/12	6/9	6/36	6/60	
14	Alagurthevar	55	M	redness + Def.vision	C	BE	N	N	-		6/60	HM	6/60	6/12	PSCC
15	Famina	36	F	Def.vision	A	BE	TB	N		+	6/36	6/60	6/9	6/12	RNTCP
16	Amsavalli	26	F	Redness	C	RE	N	N			HM	6/6	-	-	Patient not turn over
17	Alagu	28	F	Def.vision	C	BE	SLE	Scleritis keratitis	-	-	6/24	6/24	6/36	6/60	
18	Natarajan	31	M	Redness	A	BE	N	N		-	6/24	6/36	6/60	6/60	PSCC
19	Anbuselvi	40	F	Def.vision	C	BE	SLE	N	-	-	6/24	6/36	6/24	2/60	
20	Pondy	35	M	Def.vision	A	BE	TB	N	-	-	6/36	6/36	6/9	6/9	RNTCP
21	Vadivel	33	M	Floaters	C	RE	HIV +		-	-	6/12	6/12	6/60	6/12	HAART
22	Rajeswari	23	F	Pain + photo+ Def. vision	A	BE	N	N	+	-	6/12	6/6	-	-	Ref.to vitreo retinal centre
23	Papathy	38	F	Def.vision	C	LE	N	N	-	+	6/12	6/18	6/12	6/60	
24	Dhanapal	38	M	Def.vision	A	BE	N	N	-	-	6/36	6/36	6/36	6/36	PSCC
25	Manikandan	25	M	Def. vision	C	BE	TB	N	-	-	6/36	6/60	6/9	6/12	RNTCP

S.NO	NAME	AGE	SEX	SYMPTOMS	MODE OF ONSET	LATERALITY	SYSTEMIC DISORDER	ASSOCIATED OCULAR DISORDER	VITREOUS HAEMMORHAGE	NEO VASCULARISATION	INITIAL VISION		VISION AFTER 6 MONTHS		TREATMENT & FOLLOW UP
											RE	LE	RE	LE	
26	Vijaya	35	F	Redness	C	BE	N	N	-	-	6/12	6/9	6/36	6/60	
27	Priya	24	F	Asymptomatic	-	RE	HIV +	CMV retnitis	-	-	6/6	6/6	-	-	Patient not turn over
28	Mr.Annadurai	34	M	Def.vision	C	LE	N	Parsplanitis	-	-	6/6	6/6	6/6	6/6	Follow up & investigate for systemic disease
29	Sethu	41	M	Def.vison	C	RE	TB		-	+	HM	6/12	HM	6/12	Mantoux +ve
30	Vijay	15	M	Def. vision	A	RE	N	N	+	-	1/60	6/12	PL	6/12	Ref.to vitreo retinal centre
31	Sekar	27	M	Def. vision	C	BE	N	N	-	-	6/36	6/18	6/60	6/60	PSCC
32	Murugan	30	M	Def.vision	C	BE	SLE		-	-	6/60	6/36	6/18	6/9	
33	Geetha	22	F	Floaters	A	BE	HIV+	HIV+	-	-	6/12	6/12	6/60	HM	On HAART
34	Latha	26	F	Def.vision	A	BE	N	N	+	+	HM	PL	1/60	3/60	For vitrectomy
35	Banu	33	F	Redness	C	BE	N	N	-	+	6/18	6/18	6/18	6/18	
36	Ameer sasha	26	M	floaters	A	RE	TB	-	-	-	6/12	6/6	6/12	6/6	Mantoux +ve
37	Ravi	21	M	Asymptomatic	-	LE	HIV+	HIV+	-	-	6/6	6/6	6/6	6/6	On HAART
38	Anand	18	M	floaters	A	RE	HIV+	N	+	-	6/18	6/6	6/60	6/6	On HAART
39	Manikam	48	M	Def. vision	C	BE	N	N	-	+	6/60	1/60	6/24	6/12	PSCC
40	Chithamal	55	F	Redness	C	BE	TB	-	-	+	6/36	6/60	6/36	6/12	Cataract surgery done
41	Mohan	28	M	Def.vision	A	LE	N	N	+	-	6/6	HM	6/6	6/18	

Mode of onset:
A- Acute
C- chronic
laterality
U - Unilateral
BE - Bilateral
sex:
M- Male
F-Female

RE-Right eye
LE-Left eye

RNTCP-Revised National Tuberculosis Control Programme
PSCC-Posterior Sub Capsular Catract
Def. Vision - Defective Vision
HM - Hand movement
PL - Perception of Light